



## ***Acute Appendicitis and Its Complications***



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# Acute Appendicitis and Its Complications

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**TO MY MOTHER  
AND TO THE MEMORY OF MY FATHER**



## Preface

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Since there already exist excellent monographs on appendicitis since there are sections devoted to it in various systems of surgery and since it is presented in many textbooks written and edited by experienced surgeons another book on the same subject might seem at first glance to be entirely unnecessary. For several reasons I do not hold this opinion.

In the first place there is so far as I know no book dealing exclusively with the subject of *acute* appendicitis. The same comment can be made concerning presentations in systems and textbooks. The chronic disease is practically always included in the discussion of the acute disease. As a result the student and the casual reader are alike left with the idea that two conditions which are actually worlds apart are closely related to each other and are even part of the same pathologic process.

In the second place there has been a recent and I think an unjustifiable tendency for medical writers to dwell too much upon the improvement in the statistics of acute appendicitis. In 1947 according to the estimates of the National Office of Vital Statistics (based upon a 10 per cent sample) 5153 persons died from the disease in the United States. As long as several thousand persons in this country die each year from a disease which should have a minimal mortality and as long as thousands of others daily stand in jeopardy of their lives from it there would seem no rational basis for such a point of view. Until procrastination and purgation are conquered—when the mortality of acute appendicitis will be reduced to an absolute minimum—it would seem safer to keep most of the emphasis on the darker side of the picture.

In this same connection what has been achieved in acute appendicitis by the introduction of chemotherapy and antibiotic therapy is often given undue emphasis. For one thing the improvement in the statistics of appendiceal peritonitis is not always as great as it is gener-

ally supposed to be. Certainly the assumption that this complication of acute appendicitis has been conquered by this means is entirely unfounded. Moreover to imply even indirectly that appendicitis is no longer to be feared because agents exist which can control its complications is not only untrue it is upside down thinking. The emphasis again should be kept where it belongs. Early diagnosis, prompt operation and abstinence from purgation in simple acute appendicitis may be less dramatic than curing appendiceal peritonitis by chemotherapy but they will save more lives in the long run.

Finally and most important of all almost none of the presentations of acute appendicitis in monographs, systems or textbooks gives any concept of the grave and potentially lethal character of acute appendicitis. This would be a serious omission in my circumstance. It is particularly serious in textbooks in which students gain their first knowledge of this commonest of all surgical diseases.

This book is written from the point of view that acute appendicitis is an inherently serious, extremely urgent, potentially deadly disease. The entire presentation derives from that concept. The whole emphasis is on *acute* appendicitis to which too little attention is paid *per se*. The paradox still exists that appendiceal peritonitis develops because many physicians fail to recognize the syndrome of uncomplicated acute appendicitis, the recognition of which is by no means as simple as it is frequently stated to be. They fail to recognize it or having recognized it they fail to act energetically about it because they have the impression from what they have read and from what they have been taught that gangrene, rupture and peritonitis are part of the initial syndrome of the disease or because from the languid and unemphatic presentations they have read and heard they have gained no idea of its essential urgency.

The theme of this book then is the urgency of acute appendicitis. Its object is literally to sound the alarm. If I were to select a motto for it I should choose one on an old sun dial, a motto which unfortunately is still applicable to far too many cases of the disease. For all of us it is later than we think.

The background of the book is furnished by 6441 surgical cases of acute appendicitis which were treated at Charity Hospital of Louisiana at New Orleans in the 15½ year period ending 31 December 1945 and which I have been studying serially since 1933. Being impressed as we insurance companies with the value of statistics I have used the material statistically as such whenever it seemed wise and have com-

pared it with statistical data collected from the literature. I have chiefly used it however to validate the concept of acute appendicitis which I have just advanced and of which it has furnished ample evidence in every respect.

To review the entire literature of acute appendicitis would be a task beyond any man's abilities. I have however drawn freely from both the modern and the older writers from the latter of whom we have not yet learned all the possible lessons. The references listed at the end of the book are comprehensive but not inclusive.

I am glad to acknowledge the assistance I have had in the preparation of this book. The chapter on morphologic aspects of acute appendicitis and the section on the structure of the appendix in the chapter on anatomy were written by Dr Bela Halpert, Director of Laboratories of the University of Oklahoma Hospitals and Professor of Clinical Pathology, University of Oklahoma School of Medicine. Dr Halpert contributed the illustrations of the gross specimens in this chapter. He also read many of the other chapters and made many valuable suggestions concerning them. It is to his kindness I owe the contribution of the photomicrographs (Chapter V) by Dr Joseph M. Thuringer, Professor of Histology and Embryology, University of Oklahoma School of Medicine. I am greatly indebted to them both.

Dr Harry Nelson and Dr Donald Dieter, formerly of the Surgical Resident Staff of Charity Hospital of Louisiana at New Orleans, assisted in the analysis of the last two series of cases which comprise the statistical material as did Drs Clarence Bishop, J. C. Burns, Brook Garrett and G. W. Robbins of the present Resident Staff. Dr Dieter read the chapter on the clinical picture and diagnosis and the chapter on differential diagnosis and contributed many practical suggestions.

Unless otherwise specified roentgenograms are from the Department of Roentgenology, Charity Hospital of Louisiana at New Orleans. Dr Leon J. Menville, Director. They were photographed by Mr J. A. Meade. For the use of their files I am also indebted to Dr Meyer Teitelbaum, formerly Director of the Department of Radiology, Touro Infirmary; Dr P. A. Kibbe, Director of the Department of Radiology, Hotel Dieu; Drs Lucien A. Fortier, Norman S. Hunt and Joseph T. Brierre, who supplied material from the Department of Radiology of Mercy Hospital as well as from their private files; Dr John B. Coleman of the Department of Radiology, University of Minnesota Hospitals, Minneapolis; and Drs J. N. Ane, Frank J. Loria and Michael E. DeBakey, who permitted me to use cases from their private practice.

I have had innumerable courtesies from the Pathological Laboratories and the Record Library of Charity Hospital of Louisiana at New Orleans. Mr. C. Stayton Dempsey of the Record Library Staff was unfailingly helpful.

The charts and anatomic drawings were made by Miss Genevieve Lee of Vicksburg, Mississippi. The manuscript was typed by Miss Mildred Graham and Mrs. Ethel Bauer Ramond. My thanks are due to them all for their interest as well as for their competent work.

My sister-in-law, Elizabeth M. McFetridge, proposed the idea of this book and assisted me throughout with the work, including the collection of the statistical data. She is responsible for the editorial work on the manuscript as well as for the reference material and the indices. I can truthfully and gratefully say that I could not have written the book without her able and competent assistance.

FREDERICK FITZGERBERT BOYCE, M.D.

*New Orleans, La.  
2 January 1946*

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## Foreword

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With the widespread use of antibiotics many physicians have unfortunately come to believe that acute appendicitis is no longer a serious disease. Although the risk from peritonitis is not so great as it previously was it is hazardous to consider lightly either acute inflammatory lesions or obstructive lesions of the appendix.

Acute appendicitis is never a medical disease although many physicians and far too many laymen have the erroneous conception that acute inflammatory and obstructive diseases of the appendix are relatively simple conditions and that a minimal amount of experience is needed to diagnose these lesions and to treat them correctly. Such a conception is based entirely upon immaturity. The more experienced the surgeon becomes the more he appreciates the difficulty of making a correct diagnosis of acute appendicitis.

A monograph such as this is very timely and acceptable, especially one prepared and written by Dr. Boyer, whose experience with appendiceal disease is considerable. For many years he has been interested in this problem and has done a great deal of investigative work concerning it. His interest is exemplified by the fact that the present monograph is based upon a critical analysis of 6441 surgical cases of acute appendicitis treated at the Charity Hospital of Louisiana at New Orleans. Backed by this wide experience, Dr. Boyer writes authoritatively on a subject which still is so little understood. As he emphasizes and reiterates many times in the monograph, relatively few cases of acute appendicitis have symptoms corresponding to textbook descriptions while bizarre manifestations are so frequently present that the diagnosis is extremely difficult to make and the atypical case of acute appendicitis frequently taxes the ingenuity of the trained surgeon. This monograph, by accurate description of the bizarre types together with illustrative cases, will undoubtedly do much to elucidate further the



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problem of acute appendicitis and will be responsible for saving many lives which otherwise would be lost

Although the treatment of acute appendicitis while the inflammation is still confined to the appendix is relatively simple the treatment of the case of acute appendicitis with complications represents an entirely different problem Dr Boyce has considered the various complications in a thorough and masterful manner and has presented the clinical manifestations and pathologic changes so clearly that their detection becomes relatively easy The therapy is clearly illustrated and concisely described

An extremely valuable section of the monograph is the admonition to the medical profession entitled The Responsibility of the Medical Profession Only by continued effort on the part of physicians can the education of the lay public be sufficiently widespread to make parents consider the possibility of appendicitis when a child suffers from a stomach ailment and refrain from giving cathartics until after acute appendiceal disease has been eliminated

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*Historical Note*

It is a curious as well as a highly significant fact that it was a pathologist, Reginald Heber Fitz of Harvard who gave appendicitis its name, who described the pathologic changes and the clinical manifestations, who pointed to the proper therapy, and who said in his first paper on the subject practically everything that matters about the acute disease. That positive paper, as Loomis described it when he heard it read in 1886, is one of the real classics of medical literature.

It is hard to believe that a disease which is recognized today as the commonest of all acute surgical diseases and therefore of all surgical emergencies should have been described definitively more than sixty years ago. It seems hard to believe that appendectomy, which today is performed more frequently than any other abdominal operation, should still have been something of a surgical curiosity at the turn of the century and that the physician who advised its immediate performance in the course of an acute attack of appendicitis should have been regarded as something of a heretic. Finally, it is almost incredible that physicians should have ignored for so long the clues which, at least to the backward look, seem so very plain, and then when the proof was finally and conclusively put before them should for the most part have closed their eyes to the truth for another ten years.

The story of acute appendicitis is worth telling in considerable detail even in a book which is chiefly a clinical exposition of the disease.<sup>1</sup> It is highly instructive. It does not detract from the excellence of Fitz's work that much of it was based on what others had already done. As some one has well expressed it, he was not so much the pioneer student of acute appendicitis as the man who made the better mousetrap—though the path to his door is even yet not so well beaten as it might be.

As one looks back now upon the contributions to the subject made before 1880, it is easy to pick out and put together from the isolated observations and the mass of confusing details the basic facts about acute appendicitis as we conceive of the disease today: the site of the process in the appendix itself and not in the cecum in which it was long supposed to originate; the correlation of the structural change with



upon a protracted pain of the parts about the bowels is bad and it has been suggested entirely without proof that he himself died of appendicitis. It is also supposed that Lenculus in 1557 might have been speaking of acute appendicitis when he reported a rupture of the cecum in a 9 year old child with diarrhea which was checked by the grandmother who took counsel with other old women and prescribed astringent quince. Whether the disease was or was not appendicitis it might be noted that the therapy of the disease is frequently and disastrously conducted today upon the same general plan.

Other cases in the early literature can also be presumed to be appendicitis though without a scientific proof. Heister in 1733 described a postmortem examination upon a malefactor which he had performed in 1711 and during which he found a gangrenous appendix lying amid adhesions in a collection of pus. The finding seems to have been entirely accidental. In 1739 Meßner recorded what is generally conceded to be the first clinical instance of acute appendicitis in the medical literature. The patient a 15 year-old man had a right sided fluctuant tumor from which a pint of pus was evacuated. At autopsy the cecum was found to be covered with gangrenous patches and a large rusty pin was found in the appendix.

The earliest experimental observations on acute appendicitis were apparently made in 1739 by Hiebertuhn who observed that when the appendices of dogs were left open so that fecal matter escaped into the abdomen none of the animals lived longer than 24 hours. The significance of the observation naturally was entirely overlooked.

## THE EARLY NINETEENTH CENTURY

Following Meßner's report in 1739 increasing attention was paid to tumors of the right iliac fossa and incision at the point of fluctuation was frequently practiced but the responsibility of the appendix for pathologic processes in this part of the abdomen remained undetected. Early in the nineteenth century however appendiceal disease began to be mentioned.

In 1812 Parkinson in London reported the case of a 5 year-old child who died after an illness of two days. Postmortem examination revealed all the viscera to be healthy except the appendix it was perforated and its contents including hardened feces had escaped into the peritoneal cavity which was the site of a diffuse peritonitis. Parkinson realized that the perforation of the appendix was the primary cause of death. The next year Wegler a German working in France recognized perforation



the clinical syndrome the progressive character of the disease from incipient changes through massive suppuration gangrene and perforation with focal and then diffuse peritonitis the swifter progression when fecaliths are present in the appendix the extreme risk of purgation the cause of the fatal outcome the higher mortality at the extremes of life, and even the somewhat greater prevalence in men

All of these and many less important aspects of acute appendicitis can readily be derived from the earlier literature and to physicians of the post Listerian era which is also the era of safe abdominal surgery the conclusions are so perfectly clear that it seems odd that no one drew them sooner Yet no one did Even the great John Hunter missed them As Cope noted there was (since the great air raids on London the past tense must regrettably be used) in the library of the Royal College of Surgeons a record in Hunter's own hand of an autopsy performed by him on the body of a colonel who died with clinical symptoms of intestinal obstruction but in whom he found only a perforated appendix with generalized peritonitis

#### EARLY OBSERVATIONS

Although Galen's anatomic descriptions are the most complete of ancient times it is doubtful whether any of the physicians of his day (c. 130-210) completely dissected the human body At any rate Galen did not mention the appendix This organ first appears in the anatomic literature in 1522 in the writings of Berengarius Carpus Professor of Surgery at Padua and Bologna as a certain additamentum at the end of the cecum Apparently this was an original observation The appendix was first described by Vesalius in 1543 and by Fallopius in 1561 In 1561 Vidus Vidius described its structural coats Fallopius seems to have been the first to compare it to a worm Pare some years later called it the apophysis Thenceforth several authors mentioned concretions and other foreign bodies including worms in the appendix and Santorini in 1724 advanced the curious notion that the chief function of the organ was to serve as a nest for round worms where they might be preserved and cherished and thus kept from escaping into the general intestinal tract

Examination of Egyptian mummies has disclosed several instances of nonacute inflammation of the appendix as well as one instance of appendicitis with perforation and peritonitis in the body of a royal princess It is generally supposed that Hippocrates may have been describing the disease though not as such when he wrote that suppuration

scribed the possibility of an operation might be conceived some day perhaps this result will be reached.

Mezner unfortunately was not any too certain of the correctness of his views and had unfortunately said so in his contribution. As a result in spite of the soundness of his concept of both the pathologic process and the therapy of acute appendicitis and in spite of the fact that the solution of the problem of the disease thus lay ready to hand his convictions were richly overturned and the knowledge of appendicitis was retarded for another sixty years. Husson and Dence pupils of the great Dupuytren took issue with him with Dupuytren's full approval. Meniere also disagreed with him and Joliet in a work devoted exclusively to surgical affections of the intestinal tract did not even mention the appendix. Finally Dupuytren himself in his *Leçons Orales de Cliniques Chirurgicales* published in 1833 flatly denied any causid relation between right sided iliac tumors and disease of the appendix. He thus furnished another of the instances sadly frequent in the history of medicine in which a distinguished man who could brook no opposition to his own ideas kept a brilliant younger man from making a discovery of far reaching importance to all mankind.

James Copland in 1831 in his *Dictionary of Practical Medicine* first distinguished between inflammations of the cecum of the pericecal tissue and of the vermiform appendix. He stated that inflammation of the appendix might give rise to very serious affections in the cecal region and mentioned appendiceal mortification from foreign bodies with resulting fatal peritonitis though he did not believe that such inflammation could ever cause localized suppuration. In 1837 and again in 1839 John Burne physician to the Westminster Hospital concluded that inflammations of the cecum were the most frequent but least serious affections of the right iliac fossa that perforation of the appendix held second place and was most serious and that perforation of the cecum ranked third. Thus one more observer recognized the existence of two distinct diseases in the right iliac region.

In America in addition to the case reported by Prescott in 1815 and already mentioned Wolcott Richards of Cincinnati a graduate of Yale in 1837 reported a case of perforation of the appendix confirmed by autopsy in a man 35 years old. The following year Edward Hallowell of Philadelphia reported a most instructive case of a kind still very frequent and still entirely typical. A 9 year old child with pulmonary tuberculosis developed a pain in the right iliac region. A physician without seeing her (this would be the modern telephone prescription) prescribed a purgative which acted violently. Twenty four hours later the child pre-

of the appendix as the cause of death in an 18 year old youth though he believed that the disease had originated in the cecum which was gangrenous what he described as biliary calculi changed by the action of intestinal secretions probably were fecoliths

Prescott in 1815 was apparently the first American physician to report on acute appendicitis His patient who had had recurrent attacks of right iliac distress had a sudden agonizing return of the pain after a self administered enema Symptoms of acute peritonitis promptly followed and death occurred 18 hours later At autopsy the appendix was found gangrenous and perforated Meier commented twelve years later

In my opinion the fecal matter accumulated in the appendix which then dilated little by little becoming first inflamed then gangrenous and finally perforated The earliest symptoms appearing in the form of colic are probably accounted for by the inflammation and distention of the appendix its rupture occasioned the effusion which was responsible in turn for the peritonitis The perforation was determined or at any rate hastened by the patient's exertion in taking an enema since it was at this moment that the intense pain began and immediately afterward the peritonitis set in

Except that the enema itself rather than the exertion of taking it was probably responsible for the rupture one could scarcely ask for a better description of both the clinical course and the pathologic process of many a fatal case of acute appendicitis

Meier's paper which was published in 1827 contained the record of 5 cases of acute appendicitis all of which had been collected within a short space of time and 2 of which had been observed by the same physician These facts suggested to the author that if such affections were not more frequently observed it was because they were being overlooked at autopsy as well as during life In this paper is the history of what seems to be the first case of acute appendicitis which was diagnosed antemortem When Meier's friend Monsieur Sevestre was called in to see a second patient he was able to state positively that the appendix was affected so much did the symptoms resemble those in the first case which had struck him forcibly at the time as characteristic

Meier also described the recurrent abscessed type of disease previously known as iliac passion and by comparison with the cases reported in his paper showed that in this type the appendix was also the causal factor Finally he suggested—although in a footnote—If it were possible indeed to establish the diagnoses of these affections in a certain and positive manner and to show that they are always entirely circum-

scribed the possibility of an operation might be conceived some day perhaps this result will be reached.

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sented a distinct tender tumor in the right iliac fossa tympanites, and dark vomit. She died 48 hours after the purgative had been taken and postmortem examination revealed, in addition to pulmonary tuberculosis and tuberculous lesions in the intestine a perforated appendix and generalized peritonitis. The author pointed out that the tuberculous process had probably caused softening and later ulceration of the appendiceal coats and added that the perforation was minute the spill was small and acute symptoms developed only when they had been induced by purgation.

Following Dupuytren's destructive criticism of Mehier's work interest in appendicitis declined in France while it was rising in Germany. In 1830 Goldbeck at the suggestion of Puchelt of Heidelberg wrote his graduation thesis on the subject of the relation of the cecum to iliac abscess. In it he coined the name perityphlitis by which the disease continued to be known for many years thus as Kelly and Hurdon observe diverting attention from the real source of evil the appendix and clogging the wheels of progress for more than half a century by leading inquirers on the subject astray. On the other hand although he had no idea of the significance of his observations and frankly said so Goldbeck's description of the clinical syndrome of iliac disease was entirely correct.

Albers in 1838 further confused the nomenclature by introducing another misleading term typhlitis which he proposed should be used for inflammation of the cecum with Goldbeck's term reserved for pericecal disease. Twenty years later Oppolzer crowned the confusion by introducing the term paratyphlitis for inflammation of the connective tissue behind the cecum reserving perityphlitis for inflammation of the peritoneal coat of the cecum and appendix and typhlitis for intrinsic disease of the two organs.

The outstanding contribution on the subject from the German universities was published by Volz in 1846. It ranks with the earlier contribution of Mehier. Volz listed 38 cases several of them personally observed in which the symptoms of intra-abdominal inflammation were followed by perforation of the appendix as the result of fecal concretions. He doubted that except in the presence of tuberculosis and typhoid fever perforations ever occurred in the absence of concretions. He pointed out that perityphlitis was never primary and suggested that it was always secondary to inflammation of the appendix. He distinguished between the nonperforative and perforative types of disease and divided the perforative variety into localized abscess and generalized peritoneal inflammation. He stated that inflammation of the appendix was the most

frequent source of peritonitis and added firmly that if perforations of that organ were not being found it was merely because they were not being looked for. He objected to the antiphlogistic treatment of right iliac disease then in vogue as irrational and brutal and trusted that the day would come when appendicitis would be treated by rest (secured by opium) on the same principle that a broken leg is treated by immobilization. In these views he antedated Murphy and Ochsner by more than fifty years.

In 1556 Lewis published in *The New York Medical Record* 17 collected cases of disease of the right iliac fossa including abscess and other diseases of the vermiform appendix consequent upon the lodgment of foreign bodies. In 1558 Howard of Montreal reported a case of appendicitis as an instance of an affection which although not very uncommon is yet so infrequent that more than one example seldom occurs in the practice of a single individual at least in the cities the size of Montreal. The next year however Leudet wrote that for three years he had examined the appendix at every autopsy in his own hospital service and had concluded that Perforation of the ileocecal appendix is in itself more common than all other perforations of any part of the intestine whatever it at least equals in frequency all perforations of the digestive tract taken collectively.

In all according to Kelly and Hurdons tabulated collection 141 cases of appendiceal disease confirmed by autopsy were reported in the world literature between 1759 when Mestriars case was put on record and 1860. Of these 31 were reported from France 27 from England 49 from Germany 25 from the United States 4 from Canada and 2 from Italy.

#### EARLY THERAPEUTIC OBSERVATIONS

After 1860 therapy assumed an increasingly important place in discussions of appendicitis. Occasional records of the blind evacuation of pus from the right side of the abdominal cavity had been found in the literature from very early times and by the first quarter of the nineteenth century when Dupuytren and his school were practicing incision of fluctuant tumors of the right iliac fossa was quite frequently carried out. Blackadder in 1824 and Meier in 1827 had suggested the possibility of surgery for appendicitis but Hinecock in 1848 seems to have been the first to operate on an appendiceal abscess knowing what it was. His patient a woman 30 years of age had had a stormy pregnancy complicated by nausea throughout. In the fifth month following a violent right sided pain which was treated by opium calomel saline purgatives

and enemas she delivered a premature child Hancock first saw her on the eighth day of her illness On the tenth day he incised a mass in the right iliac fossa and the patient eventually recovered after the discharge from the wound of two fecal concretions the appearance of which convinced Hancock that the primary source of the trouble was in the appendix In 1856 Lewis in a discussion of the 47 cases just mentioned suggested the propriety of opening collections of pus in the right iliac fossa in cases of threatening urgency even if fluctuation were absent Hancock it should be noted had used incision as a treatment of desperation If fluctuation were waited for Lewis argued many patients would lose their chances of salvation for they would be dead before it occurred

In 1867 Willard Parker of New York reported 4 cases of suppuration in the right iliac fossa treated by incision and drainage in 1 instance before fluctuation He counseled the general adoption of this plan of treatment on the basis that what nature provides in certain cases operation can provide in all He had come to this point of view after witnessing a postmortem examination on the young daughter of a personal friend The girl had died of appendiceal disease and her death convinced Parker that in the next case of the kind he was called upon to treat he must operate between the time of the walling in of the abscess and the breaking down of its wall with subsequent diffuse peritoneal inflammation He therefore recommended operation between the fifth and twelfth day of the disease as practicable safe and justifiable These arguments many have pointed out were precisely the same as those Hancock had advanced unsuccessfully twenty years before but they won acceptance at this time probably because immediately afterward Lister began to preach the principles of antiseptic surgery and the surgical risk to life inevitable in Hancock's day was forever eliminated

Burchard in 1880 on the basis of 4 fatal personal cases of acute perforative typhlitis suggested that by a timely interference there is a reasonable hope of saving a certain proportion of lives Lawson Tait in 1881 announced that thereafter he expected to drain any peritonitis of whatever origin feeling that the treatment would prove eminently successful if the operation be not deferred until the patient is moribund Immediately afterward however Noyes wrote despairingly of the treatment of appendicitis because the difficulty of certainty of diagnosis in cases without circumscribed collections of pus would stand as an almost unsurmountable obstacle In 1883 Samuel Fenwick wrote of the rare disease which his of five years attracted the attention of practitioners and which often presents considerable difficulty in diagnosis namely inflammation of the vermiform appendix Incision and

drainage he did not consider a satisfactory method of treatment and he proposed theoretically that it would seem to be much better if we could cut down directly upon the appendix as soon as the diagnosis was tolerably certain tie it above the seat of the perforation and remove from its neighborhood any concretion or decomposing material that might be the cause of irritation. Yet a surgeon who could write these words still talked about diseases of the cecum as the chief cause of pathologic processes in the right iliac fossa.

Mikulicz of Krakow also recommended operation for perforation of the appendix at about this time. In reporting a case of appendicitis in which drainage had brought temporary relief and in which five perforations had been found at autopsy he stated that if he had only searched far enough he would have found the openings and could have removed the appendix and closed the cecum by suture. I should like to remark also he wrote that in any peritonitis the original cause of which is not clear the region of the cecum should be investigated and a possible perforation of the appendix considered. Shortly afterward Kronlein performed the operation Mikulicz had advised finding a perforated gangrenous appendix with two fecal concretions. Operation was not performed until the third day of illness and the patient a 17-year-old boy did not survive.

Deliberate interval operation for appendicitis although suggested by Blackadder in 1524 was first performed by the English surgeon Charter Symonds in 1853 on the suggestion of a Doctor Mohamed who died before the operation was done. Symonds excised the fecal concretion and instituted drainage but did not remove the appendix.

So called typhlitis and perityphlitis carried a very high mortality. There were only 3 recoveries in the 47 cases collected by Lewis in 1858 and in the 67 cases collected by W. T. Bull in 1873 there were 32 deaths 48 per cent. These were practically all nonsurgical cases. In 1882 however Noyes who had written so gloomily about the difficulties of diagnosis in appendicitis collected 100 cases treated surgically 80 of them since 1867 (the year of Willard Parker's contribution) in which the death rate was only 15 per cent the inclusion of a case of carcinoma and another of tuberculosis makes the mortality in the series seem higher than it actually was.

#### THE CONTRIBUTION OF REGINALD HEBER FITZ

Thus then was the status of disease of the appendix when in 1886 Reginald Heber Fitz Shattuck Professor of Pathological Anatomy at



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under the appropriate heading (p. 107) included the various forms of acute appendicitis exactly as they are recognized today beginning with the inflammatory process involving the appendix proper and ending with such complications as phlegmonitis with abscess of the liver. The clinical discussion was equally sound and is still correct. Pain was the most frequent symptom with nausea and vomiting next in frequency. Constipation was observed often diarrhea seldom and disturbed micturition occasionally. Fever was present in only 35 cases and was not usually very high. If physicians had remembered Fitz's observation in this regard rather than Murphy's later insistence upon fever as one of the cardinal signs of the disease a great many lives would have been saved over the course of the years.

Fitz had observed from other autopsies not included in the reported series that attacks of acute inflammation of the appendix must occur with far frequency without giving rise to characteristic symptoms or indeed to any symptoms at all. He therefore emphasized the possible latency of the disease sometimes until it was too late to save the patient and suggested that such vague diagnoses as indigestion diarrhea constipation bilious attacks colic gastritis enteritis gravel ovaritis congestion of the womb and the like may not infrequently conceal the existence of an inflamed appendix. Rectal examination was recommended as a method helpful in early diagnosis. The differential diagnosis was discussed in detail including the diagnosis of typhilitis under which term and diagnosis inflammation of the appendix had hitherto been concealed.

Taking up the question of diffuse peritonitis which he correctly described as a complication and not an integral phase of the original disease Fitz pointed out that an attack of acute appendicitis could resolve spontaneously but that resolution was not likely to be complete until long after an operation could be helpful. Another reason advanced for not waiting for resolution was the possibility of recurrent attacks of inflammation during the period of delay. If a cathartic or enema were demanded by the patient or his friends he stated bluntly it should be remembered that such medication is the means in many cases of exciting an immediate generalized peritonitis.

Rest and opium were advised as the proper method of treatment with operation if improvement had not occurred within 24 hours. Commenting on Parker's selection of the fifth day as the time of election for operation Fitz pointed out that many patients died of peritonitis long before that time and said: Hence if indications for operating justified the election of a date as early as the fifth day they still more justify

the Harvard Medical School read before the Association of American Physicians the paper with which the modern knowledge of appendicitis begins and which includes most of the modern knowledge of appendicitis. It had been repeatedly pointed out prior to this time as has been noted that disease in the right iliac fossa could be and often was located in the appendix and not in the cecum and operation for appendicitis had been advised and undertaken. Very few observers however had been bold enough to state that appendiceal disease was the predominant cause of right iliac disease and that removal of the appendix was the correct and only possible method of treatment. Furthermore no single person had had at one and the same time the correct concept of the disease and enough cases to prove his theory or had spoken loudly enough and frequently enough to make his voice heard by an unlistening and unbelieving profession.

In his paper entitled Perforating inflammation of the vermiform appendix with special reference to its early diagnosis and treatment Fitz supplied all of these lacks at one and the same time by the presentation of 257 cases of the disease which he had personally studied at post mortem examination. After outlining the long confusion between disease of the appendix and typhlitis he proposed to call this disease of the appendix appendicitis. The term did violence to the feelings of the classical scholars in his audience and still does violence to the feelings of the few classical scholars left in the medical profession but he preferred it to the term appendicular peritonitis which had been suggested by With because appendicitis is the *primary* disease and peritonitis the *secondary* consequence. That distinction regrettably is not always remembered even today.

Among Fitz's 257 cases males outnumbered females almost 4 to 1. Of the total number of patients 10 per cent were under 10 years of age and 11 per cent were over 40. The highest incidence 38 per cent was in the second decade and the next highest incidence in the third and 76 per cent of all cases occurred before the thirtieth year. 28 patients 11 per cent had had one or more previous attacks before the last and fatal attack. Foreign bodies or dried fecal masses were present in 60 per cent of the cases and external violence was a possibility in 19. Perforation was due to tuberculosis in 8 cases and to typhoid fever in 3. Indiscretions in diet and the use of domestic remedies were regarded as responsible for some at least of the 9 cases of diarrhea and 6 of vomiting and the evil influence of purgation on the progress of the disease was stated without equivocation.

The description of the pathologic changes which is quoted in full

McBurney is chiefly remembered today for his description of the point of greatest tenderness in appendicitis (p. 125) which is almost universally misquoted and for his description of the muscle splitting, or gridiron incision for appendectomy (p. 263) both of which still bear his name. He described the incision in a paper published in the *Annals of Surgery* for July 1894 but later it has recently come to light acknowledging the priority in this respect of J. J. McArthur of Chicago. In the spring of 1894 Dr. McArthur applied for space on the program of the Chicago Medical Society to present an original contribution concerning a new method of incising the abdominal wall especially applicable to appendectomy. He was assigned a place on the June program but because he was the last of a long list of speakers he agreed to postpone his presentation until the first fall meeting. McBurney's description of the gridiron incision was published the following month (July 1894). On 24 August McArthur sent him a copy of his unread paper and 10 October 1894 McBurney, who had been away from home in the interim, wrote him acknowledging his priority. Properly therefore the incision should be called the McArthur-McBurney incision.

McBurney made other contributions of value to the subject of acute appendicitis. In the first paper of his series 'Septic peritonitis following perforation of the vermiform appendix' he clearly differentiated between cases operated on early in the inflammatory process and cases operated on late which fall into a totally different category. He pointed out the frequent difficulty of determining whether or not the appendix has ruptured—a difficulty which still exists but which is not always emphasized as it should be—and declared that prompt operation should be the accepted mode of treatment. All acknowledge he wrote that every case of appendicitis may so far as the cleverest observer can tell have to pass by many very dangerous obstacles before reaching the smooth water of a comfortable abscess. For my part I would endeavor to secure safety early before reaching the rapids rather than trust to finding my way with my eyes blindfolded through a dangerous passage.

In 1900 George R. Fowler of New York described the position which now bears his name. His assistant (and son) Dr. Russell S. Fowler had observed that patients with peritonitis who were placed in a semi-upright position after operation to prevent the aspiration of vomitus were apt to have a milder course than patients who were placed supine. Fowler therefore suggested that this position which required elevation of the head of the bed for 12 to 15 inches should be used routinely in appendicitis to facilitate the passage of septic fluids from the general peritoneal cavity to the pelvic cavity where they could do comparatively little

the choice of the third day. He added that an act opening the abdominal cavity which twenty years ago might have added to the risks of the patient at the present time if properly performed might be confidently expected to reduce them very materially.

As the result of his analysis of the 257 cases in his series Fitz arrived at the following conclusions which he remarked with very good reason seem warranted:

- 1 The vital importance of the early recognition of perforating appendicitis is unmistakable
- 2 Its eventual treatment by laparotomy is generally indispensable
- 3 Urgent symptoms demand immediate exposure of the perforated appendix, after recovery from shock and its treatment according to surgical principles
- 4 If delay seems warranted the abscess which forms should be incised as soon as it becomes evident

It is impossible to overestimate the importance of a paper in which the most frequent of all surgical diseases was not only raised to its proper place but was at the same time correctly described from every aspect including the therapeutic. Loveland in fact writing in 1937 remarked that surgery had not yet caught up with this contribution. If the reader of this book does not go beyond this point he will have learned from a contribution now more than 60 years old more of practical value about acute appendicitis than he can learn from a large number of modern texts.

#### THE HISTORY OF APPENDICITIS AFTER FITZ

For the sake of completeness a few additional facts concerning the history of appendicitis should be set down in this historical note. Much of the credit for spreading the early gospel of acute appendicitis belongs to Charles McBurney, Surgeon in Charge at the Roosevelt Hospital who in the 10 year period beginning in 1888 published at least one paper a year on the subject. Today that output would not be particularly important. Then it meant that one of the most distinguished surgeons in the country had taken up the cudgels for a disease which the profession in general was still far from accepting and of which most of them still knew practically nothing. Indeed Murphy wrote in 1904 that in no procedure in surgery had the battle been so fierce and so continuous and the statements of uninformed men so personal so galling and so unjust as in the contest for and against early operation in acute appendicitis.

Two recent contributions to acute appendicitis are of outstanding importance. The first is the extensive experimental and clinical investigation of obstructive appendicitis which is still being conducted by Wangensteen and his associates and which carries to its logical conclusion the clinical description published by the late Wille of Edinburgh more than twenty five years ago. The second is the extraordinarily well sustained and fruitful campaign of public education in Philadelphia at first initiated singlehandedly by Dr. John O. Bower and later taken over by the Philadelphia Medical Society and more recently by the Pennsylvania State Medical Society (p. 115). The treatment of appendicular peritonitis, pylephlebitis, and other complications by chemotherapy and antibiotics concerns the complications of acute appendicitis, not the disease itself, and should not properly be listed as an advance in the treatment of primary appendicitis.

### CONCLUSIONS

As Hovster has succinctly put it: Anatomically the vermiform appendix has been known since the sixteenth century, pathologically it was recognized in the eighteenth century, clinically it is the product of the nineteenth century. The task of the twentieth century, though as yet it has scarcely been begun, is equally clear: to reduce the mortality of acute appendicitis to the negligible figure to which it can be reduced by the application of two simple principles: (1) prompt operation early in the disease, and (2) absolute abstinence from purgation. The application of these principles is the ultimate responsibility of the physician. He cannot absolve himself by the facile excuse that the patient was seen too late to be saved. In this era of preventive medicine it is part of the duty of the medical profession to teach the potential victims of acute appendicitis to come in time and to refrain from wrecking their chances of salvation by taking purgatives before they come.

<sup>1</sup> Although inflammation of the appendix was not called appendicitis until Fitz introduced the term in 1856, the modern nomenclature as a matter of convenience I used throughout this chapter.

The very complete bibliography in Kelly and Hurdman's *The Vermiform Appendix and Its Diseases* makes it unnecessary to repeat here references to the early literature of appendicitis.

harm and whence they could readily be removed by drainage. The value of the Fowler position was probably never as great as it was originally supposed to be and the necessity for its use has been largely eliminated by other more modern methods of treatment but it was undoubtedly very useful in the early days of appendicitis. By the irony of fate and in spite of his own extensive experience with the disease—by 1894 he had personally operated on 200 cases—Fowler died in 1906 of acute appendicitis.

In August 1904, John B. Murphy advocated the treatment of spread ing peritonitis by a plan suggested independently in October of the same year by A. J. Ochsner. Both surgeons proposed in effect that in such cases intra abdominal manipulations be limited and that the patient be treated in a sitting posture with the body at 35-40°. Oral feedings were to be withheld and from 4 to 12 quarts of saline solution were to be administered per rectum every 24 hours. This plan is an extension of the rest and opium treatment advocated by Volz in 1846 and by Hilton in 1876.

Certain important considerations concerning Murphy's and Ochsner's papers should be stressed. Neither author though both are often so maligned advocated the so called expectant treatment in the early stages of acute appendicitis. Ochsner indeed specifically stated that it should not be used during the first 36 hours of illness. Both authors recommended it for the treatment of peritonitis which is a complication of appendicitis and which was so recognized by them both. Ochsner furthermore particularly emphasized that if this treatment were employed the prohibition of oral feeding should be absolute. Mere limitation was not enough. He also pointed out that cathartics and enemas were equally dangerous and should never be employed. The condition peritonitis which I have just described he wrote is in itself the result of the administration of food and cathartics. Had these patients received neither food nor cathartics from the beginning of their attack the condition would never have advanced to this dangerous point. Ochsner's final conclusion still remains to be put into effect generally. The laity should be taught to stop feeding and giving cathartics to patients suffering from intra abdominal diseases. The continued death rate from acute appendicitis shows that the laity has not yet been so taught.

The vigorous campaigns for prompt surgery in acute appendicitis and for the withholding of purgatives which were carried on in the United States by John B. Murphy and in England by Lord Moynihan of Leeds are landmarks in the modern history of the disease. The concept of this particular book as a matter of fact derives from their work.

left and then passes over the cranial limb from which the small intestine originates and toward the right. The cecum which was previously hidden between the developing coils of small intestine thus comes to be above them. As rotation continues the colon, cecum and appendix are pushed upward and by the tenth week lie just beneath the liver and near the midline. By the end of the twelfth week the appendix is usually distinct from the cecum.

Between the fourth and seventh months of intra-uterine life the cecum usually descends from its subhepatic location to its final position in the right iliac fossa. The rate of descent varies and the descensus is sometimes not completed before term. The position and form of the adult cecum and appendix are determined by the time at which they come into contact with the posterior abdominal wall. They will remain comparatively free if during their descent other coils of intestine remain interposed between them and the abdominal wall. If on the other hand coils of ileum are displaced early so that the cecum and appendix come into contact with the posterior abdominal wall before or early in the course of their descent the posterior surface of the cecum is likely to fuse with the abdominal wall at a comparatively high level and any further descent is likely to be accompanied by rotation of the cecum to the left or toward the median line. In a small proportion of cases variously estimated at from 2 to 6 per cent the cecum and appendix are permanently arrested in their subhepatic location. During the process of rotation the appendicocolic junction becomes shifted in an upward and posterior direction and the appendix depending upon the length of its mesentery points ventrally, dorsally, cranially, caudally, medially or laterally.

Primary differentiation between the cecum and appendix as has been noted begins after the seventh or eighth week of embryonic life just after the transient process at the cecal tip has disappeared. At birth a secondary differentiation takes place in the cecum and colon especially the former which gives rise to their characteristic sacculization. As intestinal contents begin to pass through the ileocecal junction intraluminal pressure causes the longitudinal muscular coat to separate into three strips (taeniae) between which the inner coats bulge out in pouches. The separation of the longitudinal muscle into strips does not involve the appendix but the three strips converge toward its root to form its longitudinal muscular coat.

The appendix is an involuting organ with a relatively higher development in the fetus than in the infant and in the infant than in the older child and the adult. In the newborn child the appendiceal is compared



## ( II )

### *Embryology, Anatomy (including Anomalies) and Physiology of the Appendix*

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#### EMBRYOLOGY

Kelly and Hurdon's description of the embryonic and fetal development of the cecum and appendix which was based on a study of 54 human embryos in the collection of F P Mall and Max Brodel is substantially as follows

At the end of the fourth week of intra uterine life the intestinal canal has the form of a small loop protruding into the umbilical cord with a caudal and a cranial limb. The caudal limb lies to the left of the cranial limb and usually a little higher. During the fifth week the cecum appears on the outer posterior aspect of the caudal limb as a small bud which furnishes a line of demarcation between the small and the large intestine. Shortly afterward a minute process usually appears at the inner (iliac) side of the cecal bud in location and comparative size it bears the same relation to the primitive cecum that the adult appendix bears to the adult cecum. It disappears between the seventh and eighth weeks. Although this structure has been spoken of as an Anlage of the true appendix the general opinion is that it is merely transient.

After this transient appendix has disappeared the true appendix, which occupies approximately the same location begins to differentiate and to increase rapidly in size. The intestinal tract begins to recede into the peritoneal cavity by the end of the seventh week and becomes completely intra abdominal by the eighth or ninth week.

There is not complete agreement about the time at which these events occur. According to some authorities it is not until the tenth week of embryonic life that the abdomen enlarges at a rate parallel with that of the abdominal viscera and the abdominal organs are withdrawn into it.

As development continues the small intestine increases in length more rapidly than the large intestine. As a result the cranial and caudal limbs of the original intestinal loops begin to rotate. The caudal portion from which the cecum originates swings a short distance around to the

left and then pass over the cranial limb of a which the small intestine originates and toward the right. The cecum, which was previously left in between the developing coils of small intestine, thus comes to lie above them. As rotation continues, the coils of cecum and appendix are pushed upward and by the tenth week lie just beneath the liver and near the median line. By the end of the twelfth week the appendix is usually detached from the cecum.

Between the fourth and seventh months of intrauterine life the cecum usually descends from its subhepatic location to its final position in the right iliac fossa. The rate of descent varies and the descent may be more or less completed before term. The position and form of the adult cecum and appendix are determined by the time at which they come into contact with the posterior abdominal wall. They will remain comparatively free if during their descent other coils of intestine remain trapped between them and the abdominal wall. If, on the other hand, coils of ileum are displaced early so that the cecum and appendix come into contact with the posterior abdominal wall before or early in the course of their descent, the posterior surface of the cecum is likely to fuse with the abdominal wall at a comparatively high level and any further descent is likely to be accompanied by rotation of the cecum to the left or toward the median line. In a small proportion of cases, variously estimated at from 2 to 6 per cent, the cecum and appendix are permanently arrested in their subhepatic location. During the process of rotation the appendiceocolic junction becomes shifted in an upward and posterior direction and the appendix, depending upon the length of its mesentery, points ventrally, dorsally, cranially, caudally, medially, or laterally.

Primary differentiation between the cecum and appendix as has been noted, begins after the seventh or eighth week of embryonic life, just after the transient process at the cecal tip has disappeared. At birth a secondary differentiation takes place in the cecum and colon, especially the former, which gives rise to their characteristic sacculization. As intestinal contents begin to pass through the ileocecal junction, intraluminal pressure causes the longitudinal muscular coat to separate into three strips (taeniae) between which the inner coats bulge out in pouches. The separation of the longitudinal muscle into strips does not involve the appendix but the three strips converge toward its root to form its longitudinal muscular coat.

The appendix is an involuting organ with a relatively higher development in the fetus than in the infant and in the infant than in the older child and the adult. In the newborn child the appendiceal as compared

## ( II )

### *Embryology Anatomy (including Anomalies) and Physiology of the Appendix*

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Fig. 5 Barium filled appendix arising from cecum of fetal type (from Tourlet from New Orleans)



Fig. 6 Barium filled appendix arising from inverted cecum in subhepatic position (from the University of Minnesota Hospitals Minneapolis)



Fig. 7 Barium filled appendix in medial ascending position extending almost to body of fourth lumbar vertebra (from Mercy Hospital New Orleans)



Fig 1 Barium filled appendix of unusual length Note fecaliths in lumen of appendix and diverticulum of cecum



Fig 2 Barium filled mega appendix (from Touro Infirmary New Orleans)



Fig 3 Barium filled linked appendix Note fecaliths in proximal and middle thirds (from Mercy Hospital New Orleans)

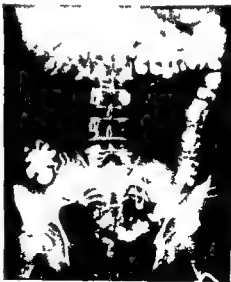


Fig 4 Barium filled knicked appendix (from Mercy Hospital New Orleans)



Fig 12 Barium filled appendix in cecal position (from Max Hight New Orleans)



Fig 13 Barium filled appendix in lateral position (from the University of Minnesota Hospital Minneapolis)



Fig 14 Barium filled appendix in subhepatic position descending lateral to colon (courtesy of Dr J N Ané)

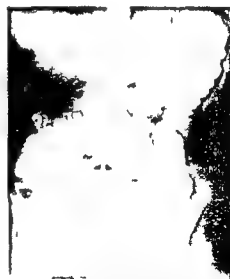


Fig 15 Barium filled appendix ascending to subhepatic position (from the University of Minnesota Hospital Minneapolis)



Fig 8 Barium filled appendix extending almost to midline



Fig 9 Barium filled appendix in subileal position



Fig 10 Barium filled appendix in pelvic position (courtesy of Dr J. V. Ané)



Fig 11 Barium filled appendix in subcecal position ascending lateral to cecum







Fig 16A Barium filled appendix and cecum both lying in midline (courtesy of Drs Fortier Hunt and Brierre)



Fig 16B Appendix and cecum visualized in fig 16A after evacuation of barium enema. Note that cecum now lies in the right lower quadrant and appendix is in subileal position (courtesy of Drs Fortier Hunt and Brierre)

to the cecal diameter is 1.3 or 1.4 in the 9 month old child it is 1.4 or 1.5 in the 8 year old child 1.6 or 1.7 and in the adult 1.6. The transition from cecum to appendix may be abrupt or the cecum may gradually taper into the appendix.

**Clinical Considerations** The developmental data just outlined have a threefold clinical significance. (1) The various positions which the appendix may occupy frequently confuse the physical findings as well as the clinical picture of acute appendicitis and so may delay diagnosis and treatment. (2) For the same reason particularly if descent has not occurred operation may be technically very difficult. (3) At least part of the more serious character of appendicitis in infants and young children is perhaps attributable to the proportionately greater length of the appendix at this age.

## ANATOMY

**Size and Shape** The appendix is a narrow tube with a blind end varying in length from 2 to 7 inches and averaging about 3 inches. Appendices 6 or 9 inches in length are not particularly uncommon.



Fig 17 Possible positions of appendix a-c descending positions d = medial positions f g ascending medial positions h ascending position i ascending lateral position j lying anterior to cecum and coiled on itself

(fig 1), and even greater lengths have been recorded such as the 12<sup>1</sup>/<sub>2</sub> inch appendix observed by Grauer (cited by Kelly and Hurdon). Extremely short appendices have also occasionally been described but tend to be of the obliterative type and therefore of no great clinical significance. The diameter of the appendix seldom exceeds 2-3 mm but may be very much larger (fig 2). There is likely to be a slight narrowing at the neck caused by the acute angle at which the appendix arises from the cecum and sometimes there is a slight dilatation at the tip. Other wise in the normal organ the lumen is of equal size throughout or gradually tapers toward the tip.

As the name implies the vermiform appendix usually has the appearance of a worm though actually its form depends upon the angle of its attachment to the cecum and upon the length of its mesentery. It may be S shaped U shaped or otherwise kinked or coiled (figs 3-4). Less often it takes the form of a straight tube. It is usually larger at the base than at the apex.

**Site of Origin** Although the appendix usually arises from the inner posterior aspect of the cecum below and near the ileocecal junction the exact point of origin may be almost any point of the cecal pouch. Treves



Fig 16A Barium filled appendix and cecum both lying in midline (courtesy of Drs Fortier Hunt and Brierre)



Fig 16B Appendix and cecum visualized in fig 16A after evacuation of barium enema. Note that cecum now lies in the right lower quadrant and appendix is in subileal position (courtesy of Drs Fortier Hunt and Brierre)

to the cecal diameter is 13 or 14 in the 8 month old child it is 14 or 15 in the 8 year old child 16 or 17 and in the adult 18. The transition from cecum to appendix may be abrupt or the cecum may gradually taper into the appendix.

**Clinical Considerations** The developmental data just outlined have a threefold clinical significance. (1) The various positions which the appendix may occupy frequently confuse the physical findings as well as the clinical picture of acute appendicitis and so may delay diagnosis and treatment. (2) For the same reason particularly if descent has not occurred operation may be technically very difficult. (3) At least part of the more serious character of appendicitis in infants and young children is perhaps attributable to the proportionately greater length of the appendix at this age.

## ANATOMY

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depends in turn upon whether the organ is straight or tortuous. The appendiceal tip may be found anywhere along the circumference of the imaginary circle. In all some ten different positions have been described but for practical purposes they may be grouped into three chief classifications: ilio-ascending and pelvic (fig. 17).

Statistical studies concerning the frequency of the various positions of the appendix are not always in agreement. It is scarcely to be expected that they would be. Observations made at operation especially through small incisions may not be accurate and alterations of position may have been caused by previous attacks of acute inflammation. The retrocecal position is often stated to be the most frequent but it has been suggested that the frequency is more apparent than real. The kinking and impairment of the blood supply of a retrocecal appendix make it more liable to inflammation and therefore make surgery more often necessary and therefore permit more numerous observations than when the appendix lies in other locations. For that matter it has been well said that whatever the position of the appendix it is strategically located for trouble.

According to Treves the appendix when examined *in situ* in the majority of cases lies behind the end of the ileum and its mesentery and points in the direction of the spleen. His postmortem studies showed that the only other position at all common was the vertical position behind the cecum with the tip sometimes in contact with the under surface of the liver.

**Contents.** The appendix is sometimes empty especially if the lumen is of small diameter and the opening into the cecum is small and slit-like or the valve is inefficient. Usually however it contains gas and liquid or solid fecal matter such as the cecum contains. Fecal concretions (fecaliths) are discussed under the heading of etiology (p. 60).

**Structure.** The appendix is neither a miniature ileum nor a miniature colon. It has a characteristic microscopic structure of its own. The inner surface is lined by a single layer of tall columnar cells many of which are goblet cells. They dip down to line the crypts of Lieberkuhn closely spaced straight tubular structures the lining cells of which produce a mucinous secretion. Beneath the epithelium is the lamina propria a delicate layer of connective tissue which contains the blood vessels and lymph channels for the epithelium. In this connective tissue there are scattered and aggregate infiltrations with lymphocytes the latter of which amount to the formation of lymph follicles. The follicles are numerous and close to each other. They vary in size. Frequently they extend through the muscularis mucosae into the

described four types of origin (1) The appendix arises in a funnel shaped opening from the lowest point of the cecal apex (fetal type fig 5) (2) It arises from the cecal apex but the base is not funnel shaped (transitional type) (3) It arises just medial to the cecal apex (4) It arises from the most medial portion of the cecal apex posterior to the ileocecal junction Wangensteen and his associates who grouped their material according to this classification classified 40 per cent of their appendices as type 1 2 per cent as type 2 52 per cent as type 3 and 6 per cent as type 4 More than two thirds of the appendices classified as type 1 were found in children under 11 years of age

The opening of the appendix into the cecum is round oval or crescentic in the great majority of cases but it may also be slitlike or irregular It varies in diameter from 0.5 to 1.5 mm

The existence of the valve of Gerlach a mucosal fold at the junction of the cecum and the appendix has been denied by many anatomists, in spite of Treves' statement that it is present in some modification in most appendices Wangensteen and his associates were able to identify it in 81.5 per cent of 526 specimens A sphincteric mechanism does not exist in connection with this valve This is borne out by the failure of these observers to find any evidence of muscle in the valve in their histologic study of adult specimens In about 20 per cent of the specimens however they observed an increase of the circular muscle on the medial or ileal side of the appendicocolic junction which suggested to them the presence of a signet ring type of circular sphincter

**Position** The appendix has no fixed position (figs 6-17) As Treves excellently expressed it the one thing constant about the position of the appendix is its inconstancy Usually the ileocecal portion of the gut is in the right iliac fossa the cecum occupying the space between the iliac vessels and Poupart's ligament If however rotation of the small intestine has been incomplete or if normal descent has not occurred (p 19) the cecum does not occupy this position and the position of the appendix changes accordingly Furthermore even if the cecum does occupy its normal position the terminal portion of the appendix may point in any direction because only its base is fixed since the cecum is a movable organ the appendix necessarily moves with it (figs 16A-B) Finally the position of the appendix depends upon the length of its mesentery and may be modified by the presence of fibrous bands and of possible adhesions to adjacent organs

The possible positions of the appendix may be visualized by regarding the cecum as the center of a circle (fig 17) the diameter of which depends upon the length of the appendix The length of the appendix

group of folds are like the meso appendix almost constantly present. The ileocolic or bloodless fold of Treves which is not always bloodless passes from the ileum to the cecum and occupies the lower angle formed by these two structures. It varies in length but is usually short and it is usually triangular though it may be quadrilateral. It consists of two leaves of peritoneum. The anterior leaf is continuous with the ventral peritoneal coat of the ileum cecum and meso appendix. The posterior leaf lies itself above on the posterior aspect of the ileum and below joins the cecum and meso appendix being reflected upward to form the upper part of the anterior leaf of meso appendix. This fold is chiefly supplied by a small recurrent branch from the appendiceal artery which sometimes must be ligated before the appendix can be removed.

The ileocolic fold passes from the ileum to the ascending colon and bounds anteriorly the ileocolic fossa which lies in the ileocolic angle between the fold and mesentery of the ileum. It is semilunar with its free (concave) margin toward the ileum. This fold which is of congenital origin arises from the mesentery a little above the terminal ileum passes in front of the ileocolic junction and lies itself on the anterior surface of the cecum. Occasionally it extends to the base of the appendix. The anterior ileocolic artery with its accompanying vein passes along the free or inner margin several lymphatic channels also pass along this margin.

Other folds which are not so constantly present include the retrocecal or subcecal fold the external parietocolic fold the internal parietocolic fold and the mesentericoparietal fold. The cecum must be lifted and rotated to demonstrate them. These various folds have suffered as Treves expressed it from a reckless and exuberant nomenclature and the descriptions in the literature have frequently done little to dispel the confusion to which the nomenclature has given rise.

In the female when the appendix and cecum are lifted up and especially when the structures are put on traction it is sometimes possible to demonstrate the presence of a thin peritoneal fold passing from the meso appendix or adjacent cecal or ileal serosa medially to the infundibulopelvic ligament. Kelly and Hurdon who admitted the existence of this fold in some cases were firm in their denial that there is any considerable vascular and lymphatic communication between the ovary and the appendix by way of it. Others take the opposite point of view.

Although Jackson's veil is not *per se* a part of acute appendiceal disease and Jackson himself specifically stated that it has nothing to do

submucosa. They frequently have clearly discernible germinal centers which are particularly prominent in children and young adults.

The muscularis mucosae is a scanty layer of smooth muscle frequently interrupted by penetrating lymph follicles. This muscle layer apparently serves to keep the mucosal surface even. Beneath the muscularis mucosae is the submucosa, a generous layer of connective tissue with occasional groups of adipose tissue cells. This layer contains the vessels supplying the mucosa. Lymphatic aggregations continuous with those in the mucosa are extensive in its inner half. External to the submucosa is the circular muscle layer, then the longitudinal muscle layer. The width of the two muscle layers around the appendix varies and the variations are even more marked in appendices of different persons.

Ganglion cells of the plexus of Auerbach are situated in the intermuscular connective tissue between the inner circular and outer longitudinal muscle layers. These cells occur singly or in groups of three to five.

External to the longitudinal layer is the serosa, which is the peritoneal covering of the appendix. The surface is covered by a single layer of mesothelial cells identical with those lining the peritoneal cavity. The width of the subserosal connective tissue shows individual variations. Lobules of adipose tissue are usually present along the border toward the mesoappendix.

The blood and lymph vessels and the nerves entering and leaving the appendix converge toward and pass through the mesoappendix. The latter is of varying width, length and thickness. The lymph nodes which collect the lymph from the appendix are usually situated beyond the mesoappendix.

A precise knowledge of the microscopic structure of the appendix supplies clues to the mechanism by which acute appendicitis develops as well as to the effects which the disease process produces within the appendix in its immediate vicinity and elsewhere.

**Mesentery.** The double fold of peritoneum which runs from the base of the appendix along all or part of its length serves as its mesentery. This fold, which is sometimes lacking, usually extends to the tip of the appendix but may not extend more than half its length. In any event, it is usually shorter than the appendix and therefore, depending upon the extent and direction of the attachment, it throws the organ into links and loops. Blood vessels, lymph channels, nerves and adipose tissue are found within the leaves of the mesoappendix.

**Folds.** The ileocecal and ileocolic folds, which form the pericecal

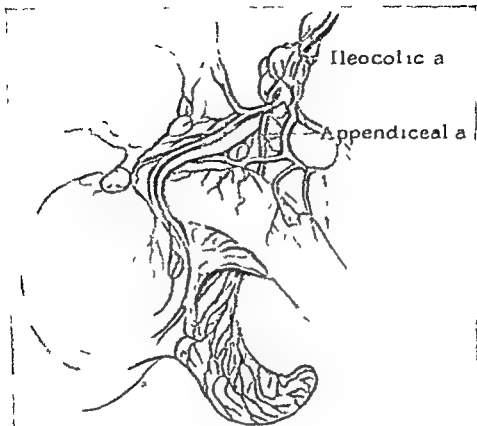


Fig 19 Diagrammatic showing of major arteries and other blood vessels and lymphatics of appendix and ileocecal region

such considerations as faulty descent and incomplete rotation of the cecum and invagination and fixation of the appendix itself by adhesions and bands. It differs from the blood supply of the remainder of the intestinal tract in that it is a terminal circulation.

The source of the vascular supply of the appendix is in the majority of cases a main appendiceal artery which arises from the ileocolic artery which in turn arises from the superior mesenteric artery (fig 19). This artery passes down behind the terminal ileum and enters the mesoappendix at its free border. It continues in an arcuate course usually at the free edge of the mesoappendix but sometimes especially if the structure contains a large amount of adipose tissue, at some distance from the edge. It may terminate at the tip of the appendix or may curve around it for 2 cm. or more before penetrating it directly.



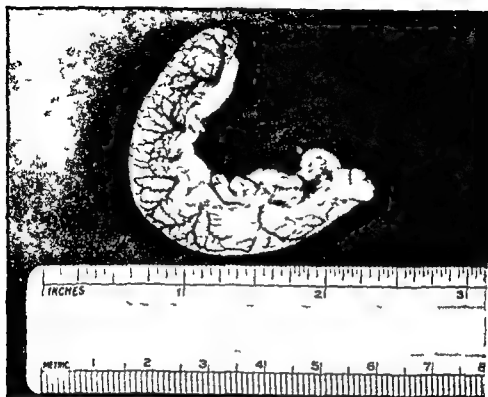


Fig 18 Acute appendicitis Note prominence of engorged and dilated vessels

with the appendix it is often found in association with acute appendicitis and operative division may be necessary before the intestine can be fully mobilized and appendectomy performed Jackson (cited by Bickham) described the fold as follows

From a point just at the hepatic flexure to three inches above the caecum there spreads from the parietal margin over the external lateral margin to the internal longitudinal muscle band a thin vascular veil in which long straight unbranching blood vessels course most of which are parallel with each other and take a slight spiral direction over the colon from the outer upper peritoneal attachment to the inner lower portion of the gut ending just above the caecum. The appendix is not implicated in any way. Coursing with the blood vessels are numerous shining narrow bands of connective tissue which gradually broaden as they go and end in a slight fan shaped attachment at various points on the anterior and inner surface of the colon. At these points of attachment the gut is held in rigid plication.

**Blood Supply** The blood supply of the appendix is out of proportion to the size of the organ (fig 18) though its efficiency is influenced by

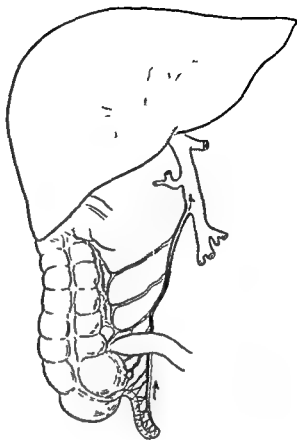


Fig 20 Diagrammatic showing of venous drainage of appendix

the border. The anastomosis of the various venous channels of the superficial and deep systems takes place either near the mesoappendiceal border or within the mesoappendix. Inside the appendix the veins generally lie close to the arterial supply. Finally they collect either into one large vessel the appendiceal vein or less often into two or three vessels which empty into the ileocolic vein in the ileocolic angle. The ileocolic vein empties into the superior mesenteric vein which in turn empties into the portal vein (fig 20).

**Lymph Supply** The lymphatics of the appendix (fig 19 p 31) are arranged in three layers: a superficial (outer) layer in the serosa, a middle layer in the submucosa, and a deep (inner) layer in the lamina propria. The occasional lymphatics of the muscular coat of the appendix drain into either the superficial or the middle system. The channels of communication between the various layers of the lymphatic supply are neither constant nor large.

The blood supply of the appendix is arranged in two layers of which the deeper is the richer there are profuse anastomoses between them. Usually the main appendiceal artery in its venous course gives off five to seven primary branches which extend at right angles to the trunk artery and are approximately equidistant to and parallel with each other. All the primary branches except the first usually divide into two encircling branches before reaching the appendix and these in turn immediately subdivide into deep and superficial layers. The superficial vessels run through the serosa and are of smaller caliber than the deep vessels which distribute themselves in the submucosa. The muscular layers apparently have no distinct blood supply. In women there is an additional blood supply from the appendicular branch of the ovarian artery.

Kelly and Hurdon list four possible arrangements of the main arteries of the appendix. (1) A single appendiceal artery supplies the entire appendix but no portion of the cecum except by way of minute anastomosing channels between them. (2) There is a multiple arterial supply. The main artery usually arises directly from the ileocolic artery and supplies the distal four fifths of the organ the remaining proximal portion being supplied by a second or even a third artery arising from the posterior ileocecal artery. The proximal branch often sends small twigs into the adjacent cecal wall but there are seldom communicating branches between the individual arteries. (3) A single artery or several arteries supply both the appendix and a considerable portion of the cecum. (4) Loops or arches are found between the main appendiceal arteries similar to those found in the mesentery. The appendiceal branches proper arise from the lower or convex side of the arches. This type of blood supply is not usual.

The capillaries of the appendix form a tortuous plexus beneath the surface epithelium between the glandular openings. This plexus drains into the venous network of the muscularis mucosae through short venous channels which run parallel to each other and to the glands. Two or more channels often unite to form a larger vessel. In the submucosa the veins converge in bundles and eventually into a few large channels which lie in the submucosa parallel to the muscular coats. The veins of the muscular coats drain partly into the submucous plexus but chiefly into the superficial plexus. The larger veins do not anastomose until they reach the mesenteric border. The veins of the longitudinal muscular coat and of the serosa drain either into large tortuous trunks which run along the surface of the appendix before turning toward the mesenteric border or into short channels which pass directly toward

always a possibility especially when foreign bodies chiefly fecoliths are present because of the mobility of its position or the position of the cecum and the effect of kinks bands adhesions and other causes of angulation. Even if fecoliths are not present the deleterious effects of obstruction are enhanced if the lumen contains fecal matter when the blockage occurs.

The presence of muscular hirtuses in which area is the mucosal tissue is in juxtaposition with the serosal tissue affords a possible explanation for the speed with which infectious processes spread when once they have developed. The terminal character of the appendiceal blood supply makes it peculiarly susceptible to interference when disease develops or when adhesions interfere with the freedom of motion which the appendix normally enjoys and which it must continue to have if it is to follow the cecum in the changes of size and position constantly undergone by that organ. Interference with the blood supply accounts for the rapid development of gangrene and the venous distribution accounts for such complications of acute appendiceal disease as pyelophlebitis and liver abscess.

The role of lymphoid tissue as a defense against acute appendicitis although frequently emphasized is doubtful particularly since the incidence of the disease is at its maximum in adolescent and early adult life the periods when such tissue is present in the greatest amounts. An explanation of the low incidence of acute appendicitis in infancy and very early childhood may be that at this period of life the great majority of appendices enter the cecum by a funnel shaped opening which does not favor obstruction at the base (Wangsten *et al*).

From a practical standpoint variations in the position of the appendix may introduce variations in symptomatology and physical findings and create considerable diagnostic confusion though many observers question the wisdom of attempting too close a correlation between position and clinical phenomena. Technical complications however are likely to be introduced at operation by variations in the position of the appendix. If it is located under the liver for instance it is often extremely difficult to remove particularly if the position has been misjudged and an ill adapted incision has been made while to visualize a retrocecal appendix it is often necessary to rotate the cecum upward and forward which is not always as easy as it sounds.

The origin of the nerve supply explains the initial pain of acute appendicitis which is usually felt in the epigastrium about the umbilicus or in parts of the abdomen other than the right iliac fossa.

Like the blood vessels the lymphatic channels pass through the muscular hiatuses in the mesenteric border of the appendix into the meso appendix. The deep system when it does not drain downward into the middle system has its own collecting channels which pass through the submucosa and converge toward the mesentery. Here they either unite with other collecting channels or enter the meso appendix independently.

In the meso appendix the collecting lymph channels course upward on either side of the blood vessels. All converge toward the appendiceal lymph nodes whose position determines the length and direction of the collecting channels, the distal channels necessarily being the longest and those nearest the cecum being the shortest. The blood vessels are enveloped in a dense lymphatic network formed by delicate anastomosing branches between the parallel lymph channels.

The lymph nodes into which the appendix drains vary both in number and in location. The reported number varies from 1 to 8. Usually the nodes are found in the ileocolic angle and usually to the left of the ileocolic vessels near the point of origin of the appendiceal artery. They have also been reported in the retro ileal, subileal and juxta-cecal areas. The collecting channels of the meso appendix empty into the appendiceal glands which empty into the ileocolic chain whose lymph channels after coursing along the mesenteric vessels drain into the receptaculum chyli. The upper continuation of the receptaculum chyli is the thoracic duct which ultimately empties into the left subclavian vein.

**Nerve Supply.** The nerves of the appendix are derived from the plexuses of sympathetic nerves around the superior mesenteric artery. From this source they run to the myenteric plexus (plexus of Auerbach) situated between the circular and longitudinal muscle bundles from which the nerve fibers are distributed to the muscular coats of the appendix. From this plexus a secondary plexus, the submucous plexus (plexus of Meissner) is derived, formed by branches which have perforated the circular muscle bundles. This plexus contains ganglia from which nerve fibers pass to the muscularis mucosae and to the mucous membrane.

**Clinical Considerations.** As will be discussed in greater detail under the appropriate headings, anatomic considerations play an important predisposing part in the etiology of acute appendicitis. The appendix is a blind tube with a narrow lumen whose contents normally teem with bacteria. Its vestigial character predisposes to trouble. Obstruction is



Fig 21 Barium filled appendix in midline pointing to left in patient with situs inversus and incomplete rotation of cecum (from the University of Minnesota Hospitals Minneapolis)



Fig 22 Left sided abscessed appendix (not shown in roentgenogram but found at operation) in patient with nonrotation of cecum (from the University of Minnesota Hospitals Minneapolis)

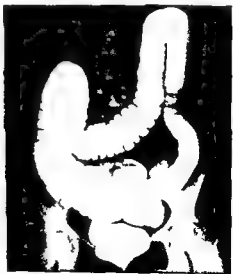


Fig 23 Barium filled left sided appendix containing fecaliths (from the University of Minnesota Hospitals Minneapolis)

## ANOMALIES

*Agensis* According to Durrh writing in 1936 ultracritical reviewers had reported up to that time only 28 authentic cases of complete agensis of the appendix since Morgagni reported the first case in 1719 and Hunter the second in 1765. Less critical writers had extended the number to 49. Durrh reported a personally observed case confirmed by postmortem examination (by five physicians) in a woman who died of gastric ulcer. Rimbo and Isky on the basis of operative evidence reported 2 cases observed in 3 years both in white males. In the first patient who was 22 years old only the appendix was missing in the other who was 18 years old both cecum and appendix were absent. Conclusive proof of agensis naturally is available only at postmortem examination when the whole bowel can be thoroughly examined.

A search of the files of the Record Library of the New Orleans Charity Hospital for the 9 year period ending 1 July 1946 revealed only 1 instance of apparent agensis of the appendix. The patient, a 17 year old girl had no vagina and was operated on through a Pfannenstiel incision to determine the state of her pelvic organs. Reasonably complete exploration showed the sigmoid on the right side but the ileocecal junction could not be found on the left side and there was no evidence of an appendix. The patient had no uterus and no tubes and there were no kidneys and ureters in the usual location though a structure assumed to be an ectopic kidney was found.

*Appendix Duplex* Since duplication of all other parts of the gastrointestinal tract is possible there seems no reason why the appendix should not be duplicated also. Actually however the anomaly is rare and many reported cases are of doubtful authenticity. Wraugh who collected 14 cases from the literature and reported a case of his own classified the 15 cases into three types: a double barreled type (5 cases), a bird type (3 cases) and a taenia coli type (7 cases). Robertson who also reviewed the literature listed four possible forms: (1) two distinct appendices, (2) an appendix with two bases and a fused tip, (3) an appendix with a single base and a bifid tip and (4) an appendix with double lumen. The most obvious explanation of appendiceal duplicity is the persistence of the transient appendix which appears and normally disappears between the seventh and eighth week of intra uterine life (p. 18). On the basis of this theory Lave (cited

sometimes only the tip extends to the left side because of such factors as nonrotation of the cecum, imperfect fixation of the cecum or excessive length of the appendix.

LeWald, who personally observed 29 cases of situs inversus viscerum, collected them in 10,000 roentgen ray examinations. One case was also observed in 35,000 examinations of recruits for the U. S. Army, and 1 in 5,000 necropsies. The incidence, based on roentgenologic examinations, would therefore be 1:1,100, but, as LeWald pointed out, this is artificially high, since many of the studies were undertaken because the anomaly was known to exist. Pol, cited by Block and Michael, found 46 cases of appendicitis, all but 2 acute, associated with situs inversus viscerum, which was total in 31 and partial in 12 instances. In more than half of the cases the pain was localized on the right side, and in only 7 was the correct diagnosis made before operation. LeWald called attention to the fact that if nonrotation of the colon is present as well as situs inversus, the appendix will be found on the right side.

J. T. Nix, Sr., observed 3 instances of left-sided appendicitis in a series of 1,215 appendectomies performed over the 20-year period ending in 1940. During this time 1 patient with situs inversus viscerum and complete rotation of the colon was also observed in the course of treatment for conditions unrelated to the appendix; one of these persons had previously been operated on for left-sided appendicitis. The writer does not specify whether the disease in any of these cases was of the acute type. It is curious that in the 6,441 cases of acute appendicitis studied at the New Orleans Charity Hospital the appendix was not located on the left side in any instance.

The finding is usually accidental, since the anomaly is perfectly compatible with health and longevity. Diagnosis, as might be expected, has been made much more frequently since the introduction of the roentgen ray. On the other hand, x-ray examination is not usually resorted to in acute appendicitis, and it is not surprising that malposition of the appendix is seldom diagnosed before operation. Some recorded instances of gangrene and perforation were found only at autopsy.

The reported cases show that pain may be on either the right or the left side, depending chiefly upon whether or not normal nerve distribution has been disturbed. Frequently the clinical picture does not differ from that of acute inflammation in the normally placed appendix. In the case reported by Block and Michael, for instance, a 26-year-old woman, 4 months pregnant, had severe griping pain in the right lower abdomen, accompanied by vomiting for 4 hours. Examination revealed a slight generalized muscular rigidity of the abdomen with extreme



by Robertson) suggested that the transient appendix may furnish substantiation of an ancestral cecal duplicity in mammals

Acute processes may occur in double appendices just as in single organs though both are not necessarily involved Schooler (cited by Waugh) who reported the first case of duplex appendix, found one organ gangrenous and perforated while the other, which was located an inch from the diseased normally located appendix was normal In Waugh's case one appendix presented purulent hemorrhagic appendicitis and peri appendicitis and the other normal histologic structure Young (cited by Robertson) on the other hand reported that one of the appendices in his case contained pus while the other had ruptured and given rise to peritonitis

Through the courtesy of Dr Joseph H Rutter of Daytona Beach who cared for the patient and Dr W H Harris of New Orleans who examined the specimen I am able to report another apparently authentic case of appendix duplex

*Case 1* The patient a white male weighing 207 pounds felt for 24 hours as if he had eaten something which had disagreed with him When Dr Rutter first saw him at the end of this time there was generalized abdominal tenderness but no rigidity examination was not particularly satisfactory because of the thick abdominal wall The white blood cell count revealed no abnormalities Twelve hours later the clinical picture was unchanged The temperature ranged between 97° and 98.5° F A few hours later nausea was present for the first time tenderness and rigidity were elicited in the right iliac fossa and the white blood cell count was 19 000 per cu mm with 70 per cent polymorphonuclear cells and 8 per cent sticks Operation revealed two gangrenous appendices The stump of the anatomic appendix could be readily handled by the usual purse-string technique but the accessory appendix was so close to the ileocecal valve that a purse-string could not be applied and a fecal fistula developed which however closed spontaneously several weeks after operation

After examination of the anatomic appendix Dr Harris made a diagnosis of acute diffuse suppurative appendicitis with mural necrosis hemorrhagic extravasation and serosal involvement Examination of the accessory appendix revealed the same findings together with involvement of the mesentery The location of the accessory appendix and its length and tubular structure seemed to exclude the possibility of diverticulum

*Left Sided Appendix and Situs Inversus Viscerum* The true left sided appendix is almost always the result of situs inversus viscerum either complete or partial (figs 21-3) It seems scarcely logical to classify under this heading cases in which only part of the organ and



Fig 21A Barium filled appendix in sub-cecal position containing BB shot (courtesy of Dr F J Loria)



Fig 21B Appendix shown in fig. 21A after evacuation of barium enema. Note that 4 of the BB shot previously shown in the appendix have passed into the cecum (courtesy of Dr F J Loria)

invalidated by the recent observations of Wingensteen and his associates which have shown that the appendix although a part of the segment of the bowel which absorbs fluid is itself able to secrete fluid and can spontaneously build up an intraluminal pressure of about 40 cm of water. Additional proof of the usefulness of the organ is demonstrated by the hundreds of thousands of cases in which it has been removed without detriment (and with great good) to the host.

The theoretic functions suggested for the appendix are matters of mere academic interest since none of them has been proved. They include the extraction of harmful substances from the cecum, the initiation of peristalsis in the large bowel, the initiation of the desire for defecation, the elimination of bacteria, the production of a lubricating substance for use in the cecum, the production of other substances including lactopeptone, hormones, and special digestive enzymes specific for certain substances. One particularly untenable idea was suggested by Keith (cited by Royster) that for complete cecal digestion all food must pass through the appendix.

That peristalsis does occur in the appendix has been shown by radiologic studies (appendiculography). Figure 21A which shows birdshot in the appendix and figure 21B which shows them in the

tenderness in the right iliac region. The patient lay with her right thigh flexed on the abdomen. At operation through a McBurney incision visceral transposition was found. The patient, curiously, had known that her heart was on the right side but had failed to mention it until the appendiceal anomaly was discovered.

In a case reported by Pool a 14-year-old boy complained of generalized pain at the beginning of the attack. Localization occurred in 48 hours. The pain was at first more severe on the left side and later on the right. Tenderness was bilateral but more marked on the right. When the limbs were alternately extended pressure over the psoas muscle caused greater pain on the right side. At operation the cecum and appendix were found in the left iliac fossa and the appendix was gangrenous. In Haddons' case in which the cecum and ascending and descending colon lay entirely on the left and the small bowel on the right the appendix lay obliquely across the left lower abdomen with the tip touching the rectovesical pouch.

**Clinical Considerations.** The anomalies of the appendix that have been mentioned are exceedingly unusual and would be of almost no clinical significance except for the well-known fact that the rarest of conditions may be just around the corner. Agenesis, as has been intimated, should not be left presumptive at operation for presumable acute appendicitis but should be diagnosed only after as careful and thorough an examination as is possible. When the appendix is missing from its normal location at operation the hepatic flexure should be explored to see if the cecum has failed to descend and the contents of the left iliac fossa should also be investigated.

Occasionally a double appendix explains the cases in which the appendix is found *in situ* at a second operation when it was supposed to have been removed at a first operation. Appendix duplex. Robertson pointed out his real medico-legal significance as was demonstrated in the Canadian courts in 1935. He therefore suggested that when a subacute appendix is found at operation in a patient with acute symptoms it might be wise to institute a further search for a second, possibly acutely inflamed, appendix.

### PHYSIOLOGY

When one speaks of the function of an organ one implies the usefulness of the structure in the bodily economy. In that sense the appendix has no known function. Like all embryonic structures it seems to exist merely to make trouble for the host. That statement is not

### ( III )

## *Etiologic Factors in Acute Appendicitis*

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The reduction of the mortality of any disease depends upon two factors the therapy employed when it develops and more important though not always as practical the measures taken to prevent its development. For a long time nothing whatever was known about the etiology of acute appendicitis and even now there are only two theories which are at all tenable the theory of infection and the theory of obstruction. Prophylaxis in the sense of actual prevention of the disease is therefore entirely impractical at this time.

One reason why the etiology of acute appendicitis has not been completely clarified is the difficulty of experimental reproduction of the disease. The appendix of the chimpanzee closely resembles that of the human subject but the general experimental use of this animal is necessarily limited. The dog possesses a cecal appendage in many ways like that of man but the differences are sufficiently great to make experimental results not precisely applicable clinically. Ligation of the appendiceal blood vessels and meso-appendix causes gangrene and is generally followed by peritonitis and death but this is not the usual mode of the production of human appendicitis. Further difficulties arise from the fact that the intestinal flora of animals and humans is different.

The various predisposing causes and etiologic factors which have been proposed for acute appendicitis are discussed in this chapter chiefly to eliminate them at least in a universal application.

### FAMILIAL AND ENDEMIC CAUSES

Apparent outbreaks of acute appendicitis have been reported in families as well as in institutions military installations and similar aggregations of persons but there is nothing to suggest a contagious or hereditary origin for the disease. Generally speaking it seems more reasonable to explain numbers of cases in the same family or group by the fact that acute appendicitis is the most frequent of all surgical diseases and that the operation of the law of averages would be likely to produce several cases in the same family or group over given

cecum furnish clear cut evidence of such peristaltic action. According to Wood when the appendix is completely filled the walls are smooth. When it begins to empty the rate of emptying being the same as that of the cecum peristalsis gives it a segmented appearance so that it looks like a string of small sausages. Peristalsis also causes it to change its position if the meso appendix is sufficiently long its action is like that of a pendulum.

Westphal (cited by Bowers) pointed out that peristalsis usually begins at the base where the musculature is normally heaviest and most active and progresses toward the tip the bulbous shape of which makes its complete emptying difficult.

*Clinical Considerations* The importance of the secretory function of the appendix in the production of obstructive appendicitis is considered in detail in the section on etiology (p 60). The fact that the organ has a peristaltic function partly explains why when disease has been initiated it often progresses so rapidly since generally speaking movement serves to disseminate infection regardless of where infection is located. The peristaltic function of the appendix also explains why purgatives which increase peristalsis have such a disastrous effect in both the infectious and the obstructive type of lesion.

<sup>1</sup>The description of the structure of the appendix was written by Dr. Bela Halpert, Director of Laboratories of the University of Oklahoma Hospitals and Professor of Clinical Pathology, University of Oklahoma School of Medicine who also contributed the chapter on morphologic aspects of acute appendicitis (p 75).

and another son and the mother were operated on for chronic disease. Three other daughters had had recurrent attacks of appendicitis but to the date of writing had not been operated on. Another daughter and another son had no abdominal complaints.

Perry and Keeler reported the history of a single New England family in which 39 cases of appendicitis had occurred. Two brothers married two sisters and there were some marriages between cousins but the families were of a high degree of intelligence with no history of nervous disorders and their environmental and dietary conditions were excellent. The sex distribution in the 39 cases was 16 males and 23 females and the incidence of the disease in the lower section of the pedigree was much more frequent than in the upper. When the portion of the family in which appendicitis was most concentrated was examined it was found that a man with an appendicitis family history had married a woman with a similar family history. The authors were of the opinion that some sort of hereditary appendiceal diathesis had been transmitted and that long large bent coiled or retrocoiled appendices were apparently hereditary characteristics though they granted that kinking and coiling of the appendix might be acquired as well as hereditary. Their conclusion was that a number of the factors which predispose to appendicitis may be inherited as irregular dominants and that in the family described the genes for several of the predisposing physical factors were concentrated in the germ plasm of one woman and her descendants one of whom married into another appendicitis family.

The instances cited represent unusually heavy groupings of appendicitis in single families but at that the arguments for a family predisposition do not seem convincing. It is possible that even such unusual concentrations of the disease within single families can better be explained by the great frequency of acute appendicitis combined with the law of averages perhaps with some assistance from the long arm of coincidence.

### SEASONAL CAUSES

A seasonal incidence has frequently been suggested as a possible indirect cause of acute appendicitis but remains unproved. It is true that some series of cases do show higher incidences during certain months of the year than during others but there is no uniformity in the distribution and many series studied from this standpoint show graphs that are practically level. The increased incidence in the spring and summer which some observers have reported has been attributed to the ingestion of such foods as berries and grapes which are seasonal at these times but

periods of time. The acceptance of the theory that the same presumably faulty diet is consumed by all members of the same family or group would imply the acceptance of a dietary origin which as pointed out elsewhere (p 47) is not generally applicable. The explanation that a family or group lives under the same environmental conditions is also not applicable no evidence exists to suggest that living conditions have anything at all to do with the development of acute appendicitis.

Although there is almost nothing in the incidence of the disease to prove a hereditary origin it has been suggested that certain families may inherit either some diminished personal resistance to infection or some similar malformation of the appendix. Too little is known at this time about the inheritance of disease to justify dogmatic statements but it seems fair to say that even if one grants the possibility of inheriting anatomic malformations of the appendix or a lowered resistance to infection it would still be necessary to explain why acute appendicitis develops in certain individuals of a family but not in others and information in regard to what students of genetics term the environmental trigger stimulus would still be lacking. It seems entirely unreasonable to assume that individuals in a supposedly susceptible family could possibly be kept from developing the disease by such measures as rest general hygiene proper food removal of foci of infection and similar measures which have been suggested but which would seem to have a most nebulous connection with acute appendicitis.

It may be of interest to cite a few of the occasional instances of apparent familial disease that are on record in the literature. Downs for instance traced through three generations a family of 22 members of whom 16 required appendectomy 10 for acute disease in 1 of the 2 cases with perforation operation was performed within 11 hours of the onset of symptoms. In every instance laparotomy revealed a band of fibrous tissue binding the appendix to the outer aspect of the cecum and causing it to be sharply kinked. Downs theory is that in this and similar cases the same congenital malformation of the appendix might be a predisposing cause of disease but would not be sufficient in itself to precipitate an acute attack.

Leckie reported a family history in which the disease was of more serious character. The grandfather died of inflammation of the bowels which in the light of later knowledge was assumed to be acute appendicitis. The father died after repeated attacks of indigestion one daughter died of appendicitis with perforation and 3 other daughters survived perforation. One son required appendectomy for acute disease.

chief the history of other attacks. Fitz seems to have shared this opinion for he wrote

In considering the symptoms of appendicitis it is to be noted that attacks of inflammation frequently occur without giving rise to any characteristic symptoms and often without suggestion of any distinct malady. The severity of these lesions found at autopsy suggest that apparently slight disturbances of digestion were overlooked or regarded as characteristic of a feeble digestion.

Opinions also differ on whether a previous attack predisposes to more serious or less serious pathologic changes in subsequent attacks. In the Charity Hospital series in which the history of previous attacks is frankly not reliable their occurrence seems decidedly to have modified the severity of subsequent attacks particularly in the patients between 13 and 39 years of age the effect in older patients was considerably less marked (fig. 73 p. 312). It is reported a 75 per cent incidence of perforation in the first attack in his series as compared with a 57 per cent incidence in patients with previous attacks. McClure and Altemeier reported that 26 per cent of 252 patients with appendicitis accompanied by perforation had had previous attacks and that in a third of that group perforation occurred within 24 hours or less of the onset of the current attack. Reid and his associates who reported that the incidence of perforation in patients with previous attacks was 42.5 per cent interpreted their statistics as indicating the risk of treating the first attack expectantly.

The present tendency to remove the appendix promptly during the first acute attack will undoubtedly mean in time that fewer and fewer patients will be seen with a history of previous attacks. The present practice of interval appendectomy the unfortunate tendency to remove the appendix for so called chronic appendicitis (p. 393) and the practice of prophylactic appendectomy in the course of abdominal surgery for other diseases will also have the same effect.

## RACIAL AND DIETARY CAUSES

*Racial Incidence* Whatever the reason there seems little doubt that ✓ appendicitis is a disease of civilization. It is frequent in Europe and Australia very infrequent in Asia Africa and the Polynesian Islands and more frequent in the United States than anywhere else in the world. It is quite true as numerous observers have pointed out in connection with other diseases as well as with acute appendicitis that not a great



as early as 1896 Edmund Andrews disposed of that theory by a statistical study of appendicitis during the grape eating months of the preceding 14 years

The increased incidence of acute appendicitis during the fall and winter months reported by other observers has been explained as due to the increased incidence of upper respiratory infections at these times. That there is a rather frequent association of the two conditions, particularly in young children is undeniable. In the Charity Hospital series 418 patients 19 of whom died had colds or other mild or relatively mild upper respiratory infections at the onset of the illness and 7 others developed acute appendicitis in the course of pneumonia. The literature also contains numerous illustrations of acute appendicitis following epidemics of upper respiratory infection. Thus Dawson reported that shortly after an epidemic of tonsillitis and other upper respiratory infections which had affected 50 of 150 men at a certain camp 4 other soldiers developed similar infections. Within 12 hours 3 of the 4 had been operated on for acute appendicitis which in 1 instance was associated with gangrene and four days later the fourth man was operated on. A little later 5 men who had been ill during the epidemic of respiratory infection also developed acute appendicitis.

That associations of this kind are anything more than coincidence no one has been able to prove. It is possible as Gray and Heifitz observed that the lymphoid tissue of the appendix may sometimes partake of a general bodily reaction and that the swelling of the follicles may even cause mild obstruction of the lumen of the appendix (p. 62).

### PREVIOUS ATTACKS

How large a part a previous attack of acute appendicitis plays in a succeeding attack it is difficult to determine. As is pointed out elsewhere (p. 103) such residua of previous attacks as intraluminal strictures and contractions; extrinsic bands and adhesions and other anatomic changes do play a part if they are present but they are not in evidence in many if not in most of the appendices removed after previous attacks.

Opinions differ in regard to the importance of previous attacks. According to Royster 98 per cent of patients with one or more previous attacks have subsequent attacks. Others set the frequency of recurrence much lower though Royster's statement is undoubtedly correct that when a patient in the midst of an acute attack states that it is his first he often means only that it is his worst and careful questioning will

duced seem to support the assumption. Thus Dawson cited the observations of a medical officer who in 4 years on a tropical island did not see a single case of acute appendicitis among natives and saw only one in army personnel. Within 3 weeks after a cold storage meat plant had been put into operation he treated 12 cases in army personnel and 7 cases among natives. McClure, in discussing Hoggins' paper on the appendix problem, stated that on the Ford rubber plantation in Brazil there were no cases of acute appendicitis in the first year of its operation, but that each year thereafter there was a progressively larger number and eventually gangrenous appendicitis was not uncommon. Over this same period the living standards of the natives had steadily improved and fresh meat and canned goods had tended to replace their former diet of fish and fresh fruit. Superficially the cause and effect relationship seems clear. On the other hand, acute appendicitis is practically unknown in the true Arctic and in such places as Abyssinia and Madagascar where the diet consists largely of meat. It may be of interest in this connection that apes in captivity, according to Short, require a susceptibility to acute appendicitis though the observation is of little practical value since no one knows a great deal about the disease pattern of apes in their native habitat.

Short, commenting on the infrequency of appendicitis in Barbadian Negroes, related it to the fact that in Barbados sugar is a staple article of food among the black race whose babies are often brought up on it. The speculation becomes less valid, however, when one recalls that America, where appendicitis is more frequent than it is anywhere else in the world, had for many years before World War II the highest per capita consumption of sugar in the world. It will be of interest to note whether any change in the incidence of the disease will follow the rationing of sugar in effect between 1942 and 1947; too many other factors enter into statistical calculations to permit even a speculative statement at this time.

The cellulose rich diet of primitive peoples, in contrast to the cellulose poor diet of civilized persons, does not seem adequate as an explanation for the infrequency of acute appendicitis in uncivilized persons though Short, who made the first observations on the cellulose theory some 25 years ago, has recently published a book on the subject with statistics brought up to date. His argument is that the roughage properties of cellulose, rather than any particular chemical virtue in it, serve to prevent the development of appendicitis. One of the chief arguments against his theory is that appendicitis occurs in nursing infants and in meat eaters who get no cellulose at all. It is of interest, however, that

deal is known about the incidence of disease in primitive and uncivilized peoples and that many statements in regard to the absence of special conditions among them are based on lack of knowledge of causes of death rather than on scientific proof. On the other hand considerable evidence does exist concerning the infrequency of acute appendicitis in more primitive peoples.

One or two typical illustrations might be mentioned. Galbreath and Irwin for instance, among 14 124 admissions over a 9 year period at the San Juan Presbyterian Hospital in Puerto Rico observed only 244 cases of all types of appendicitis only 20 of which occurred in natives. Fossen and Beocke in a 5 year period at the Central Civil Hospital in Batavia treated only 175 cases of acute appendicitis in 13,000 admissions. The incidence based on admissions was 4.5 per cent among Europeans 2 per cent among Chinese and 0.6 per cent among natives of Batavia. Loveland mentioned an army officer in India who over a period of 15 years neither saw nor heard of acute appendicitis and a medical missionary in Arabia who saw only 1 case in 20 years. Judd (cited by C. W. Mayo) during his 7 years as a missionary in China saw no case of appendicitis although Chinese become susceptible to the disease when they move to America. Weiskler (cited by Royster) observed only 2 cases of appendicitis in more than 80 000 clinic admissions at Tientsin China and one of these occurred in a nun attached to an order that served European food.

Willson Pepper who was stationed on the West Coast of Africa during World War II in a hospital on the outskirts of a large native town 100 miles from the coast stated that acute appendicitis was seen among Europeans in about the same proportion as in Europe but that he did not see a single case in an African native. American medical officers have commented on the very low incidence of appendicitis in native Philippine troops. Steiner carried out 150 postmortem examinations on natives of Okinawa ranging in age from 18 days to 95 years during June and July 1945. Most appendices were comparatively normal. One organ 1 cm long terminated in a slender fibrous cord and another was involved in an extensive acute ulcerative process of the ileum and colon which had perforated and caused peritonitis but there were no instances of acute appendicitis in the whole group of deaths 50 of which were due to noncombat conditions. Appendicitis was not mentioned at all by native physicians who were questioned as to the health of the natives.

*Racial Food Habits.* It is generally assumed that the infrequency of acute appendicitis among primitive persons is due to their food habits and changes in incidence reported when habits of civilization are intro-

make it clear that no explanation for the apparent infrequency of acute appendicitis among uncivilized peoples is now visible. One other phase of the matter however must be mentioned for it is of great practical importance at least in certain sections of the United States that acute appendicitis has become increasingly frequent and apparently increasingly severe in the Southern Negro.

In 1896 when Miles wrote his classical essay on the surgical peculiarities of the American Negro based on studies of various diseases at Charity Hospital of Louisiana at New Orleans he found only 34 instances of appendicitis for the whole preceding decennium. Twenty five cases with 7 deaths occurred in whites and 9 of which 2 were fatal occurred in Negroes. Appendicitis was then 10 years after Fitz had published his illuminating contribution still a relatively unknown disease in the South and the numbers are too small to be of statistical value but the disproportion between white and Negro subjects is nonetheless characteristic as are the disproportionate racial mortalities.

Today with enormously increased incidences of the disease in both Negro and white subjects the disproportions are still very much in evidence. For most of the almost 16-year period covered by the study of 6441 cases of acute appendicitis at the New Orleans Charity Hospital the ratio of Negro to white hospital admissions was approximately 45/55 and for the remainder (most recently) it was approximately 55/45. The Negro furnished however only about 29 per cent of the incidence of acute appendicitis over this period though his proportion of fatal cases was more than 42 per cent and the Negro mortality (7.26 per cent) was almost twice the white (4.04). In the opinion of many observers and after making full allowance for such possible explanations as greater procrustianism and more frequent purgation on the part of the Negro acute appendicitis like most required conditions seems a more serious disease in the Southern Negro than in the white subject.

*Dietary Indiscretions.* Although special articles of diet or even the diet of civilization as a whole cannot be directly impugned as the cause of acute appendicitis there is no doubt of the importance of dietary indiscretions in the disease. In some instances such indiscretions seem to serve as a precipitating factor. In the Charity Hospital series the acute attack followed dietary imprudence in 166 cases in 17 of which 10.24 per cent it proved fatal. It is well known that appendicitis often shows an increased incidence around Christmas and Thanksgiving when over eating is very frequent. Bowers related the role of dietary indiscretions to an increased secretory and peristaltic activity in the intestinal tract which may initiate a pressure distention mechanism in the appendix.

Magee observed that in the Channel Islands during World War II as food shortages increased and diet became coarser and more fibrous the incidence of acute appendicitis decreased<sup>1</sup> Aschoff mentioned the similar observation reported by Hamperl with regard to the decreased frequency of appendicitis in Russia in the years when some degree of starvation was common but was inclined to attribute it to a possible alteration of the bacterial flora of the appendix alteration in the secretions chemical changes in the intestinal contents or the absence of the pressure that the normally filled cecum exercises on the appendix and its curves

Rovster's observation that the rise of the incidence of appendicitis in England dates from 1895 which is about the time that the nation ceased to feed herself and began to import a large amount of her food is vitiated by the fact that this is also just the time that appendicitis began to be generally recognized as a disease entity In America the rising incidence of acute appendicitis coincides strikingly with the falling incidence of such diseases as diarrhea and enteritis both of which may have a dietary etiology

Racial Living Habits Lewis and Firor offered the explanation that modes of living that restrict the natural tendency to frequent and complete defecation may favor the development of appendicitis The squatting posture with thighs flexed on the abdomen which most primitive races assume for defecation tends to empty the cecum completely whereas the posture assumed by civilized persons does not The acceptance of this theory would imply the acceptance of constipation as a cause of acute appendicitis and this is not reasonable Appendicitis occurs quite as often in persons with a history of perfectly regular bowel movements as in those with a history of constipation and great numbers of habitually constipated persons go through life without ever suffering from appendiceal disease

The suggestion that aborigines who live an active outdoor life have better intestinal tone and possess greater resistance to infection than civilized persons also does not seem valid On that reasoning athletes would not develop the disease yet football players baseball players pugilists tennis players and others have been stricken with it at particularly embarrassing moments in their careers Lowered resistance in fact cannot have anything to do with the production of the disease Appendicitis is no more frequent in sickly individuals and invalids than in well and strong subjects Indeed it very often strikes down its victim in the midst of vigorous health

*Acute Appendicitis in the Southern Negro* Enough has been said to

and the number of acute cases in which they play an etiologic role is extremely small though it must be granted that the proportion of positive findings depends upon the expertness of the technique employed to identify them. It has greatly improved in recent years. A few instances will suffice to prove this point.

Bowers who reported finding parasites in 3 per cent of 185 cases of appendicitis observed no instance of coincident parasitism and acute appendicitis. Ashburn found oxyuriasis (*Intestobius vermicularis*) present in 184 of 2317 surgically removed appendices 7.91 per cent but oxyurids were as frequent in the normal as in the chronically inflamed appendix and more frequent in both than in the acutely inflamed organ. Harris and Browne found 21 instances of oxyuriasis in 121 consecutively examined appendices the association being chiefly with subacute and chronic disease and Schenken and Moss who studied 1000 consecutively removed appendices reported substantially the same observation.

In the New Orleans Charity Hospital series of 6441 surgical cases parasites were reported in only 62 patients 37 of whom were children 12 years of age and under the actual incidence would undoubtedly have been higher had all the specimens been properly studied from this standpoint. There were 5 deaths in the group all in children under 12 years of age. Parasites were also present in 5 cases all in children and young adolescents in the 158 fatal nonsurgical cases studied at the New Orleans Charity Hospital. They seem to have played the same part in confusion of diagnosis and harmful treatment as in the surgical cases.

In no case in the series so far as could be determined did the parasites chiefly round worms play an etiologic part though their presence if it was known prior to the attack obviously accounted for confusion in diagnosis and for the use of purgation in many of these unfortunate children as the following history shows.

**Case 2.** A colored child 3 years of age had been known for several months to have intestinal parasites. Six days prior to hospitalization he passed several worms by rectum and was given a vermifuge followed by castor oil. On the following day he developed severe abdominal pain and was given milk of magnesia which was not effective. He vomited twice the vomitus each time containing worms but had no other symptoms except persistent abdominal pain. The temperature was 101.6° F. the pulse rate 130 and the respiratory rate 28 per minute. The child did not look acutely ill. Physical examination revealed tenderness and moderate rigidity over the entire right side of the abdomen and bilateral rectal tenderness. The white blood cell count was 22,600 per cu. mm. with 81 per cent polymorphonuclear leukocytes.

Appendectomy, cecostomy and drainage were carried out immediately.

That the dietary indiscretion is responsible for the high mortality in these cases seems doubtful. A simpler explanation would be that the history of dietary imprudence clouds the picture diagnosis is accordingly delayed or not made at all (a history of this kind was obtained in 13 of the 158 fatal cases at the New Orleans Charity Hospital in which no surgery was done) and that purgatives often with apparent logic are used in the treatment of the supposed gastro enteritis. Willson Pepper attributed the high incidence of gangrene and perforation of the appendix in his material from the West Coast of Africa to the high incidence of diarrhea which he attributed in turn not to pelvic peritonitis or abscess but to the high incidence of parasitic disease of the intestine in a tropical country his reasoning was that the appendix once it has been involved in the diarrhea purges of the excessive peristalsis of the irritated bowel. Morrissey reporting on appendicitis in an Army Air Force regional hospital also made the point that involvement of the appendix in a patient with diarrhea leads rapidly to perforation.

**Purgation** Purgation in addition to playing an important role in the progressive pathologic change in acute appendicitis sometimes seems to initiate the attack. In 54 cases in the Charity Hospital series 5 of which were fatal acute symptoms supervened within a short time after a purgative had been taken. In every fatal case the death could be attributed to delay caused by that fact. The sequence of events in these cases is not entirely clear. Coincidence may be the sole explanation at least in some cases. That purgation can initiate acute appendicitis in a previously normal appendix does not seem likely though as Bowers experimental studies have shown it can readily initiate it in a potentially or actually obstructed organ. Another possible explanation is that a patient with mild or vague symptoms of illness or lack of well being which actually are the first symptoms of acute appendicitis takes a purgative on general principles as many persons do and forgets the premonitory troubles when the more serious ones supervene. Be this as it may in every fatal case in this group in the Charity Hospital series death could be attributed to the delay caused by the confusion introduced by the purgative.

### PARASITISM

At first glance it might seem that intestinal parasites would play an important role in the etiology of acute appendicitis because they might serve as *foreign bodies* and might supply a portal of entrance for infection. Actually the number of cases in which they are present is small.

In contrast to these doubtful cases is a case reported by Zahawi. A 10-year-old boy died in coma in a Baghdad hospital shortly after admission presumably of intestinal obstruction due to impacted ascariides. Autopsy revealed four ascariides in the appendix the wall of which was markedly thinned and atrophied from pressure by them. The writer speculated that if death had not occurred from toxemia the appendix would undoubtedly have ruptured from internal pressure. If the speculation is valid the case would seem to be one of the extremely unusual instances in which parasites played a direct etiologic role in the production of an appendiceal lesion which was not however true acute appendicitis.

The association of parasitism with recurrent (chronic) and subacute appendicitis whether by coincidence or on a cause and effect basis seems fairly frequent particularly in countries in which parasitism is of high frequency. Hamrah for instance reported that in Palestine *Indoamoeba histolytica* causes 20 to 30 per cent of all cases of subacute or chronic appendicitis.

### FOREIGN BODIES

Foreign bodies have been charged with the responsibility for acute appendicitis ever since Meisnier in 1759 recorded the first case in which a pin was found in the appendix (p. 5) and Fitz mentioned their possible role in his first paper on the subject. Curreri and Melick in a review of the literature found that foreign bodies actually observed in appendices include pins needles nails screws shot (fig. 24) bullets iron filings solder stones pebbles buttons seeds pieces of bone and wood beans oat hulls chestnuts bristles egg shell hair teeth twigs and straw.

Any foreign body if it is large enough may occlude the lumen of the appendix (p. 60) and produce the obstructive type of acute appendicitis just as any sharp body may erode or perforate the wall. Pins which most readily perforate the wall are associated with a very high mortality. Except by means of these two mechanisms however the presence of foreign bodies seems to play little or no part in the etiology of the disease. The fact that most appendices do not contain them eliminates them as a general factor.

A few of the extremely interesting cases of foreign bodies in the appendix which have been reported might be cited.

Earl reported the accidental finding by roentgenology of a BB shot in the appendix of a patient under examination for another condition.



under ether anesthesia through a McBurney incision. The appendix was gangrenous and ruptured, the cecum was acutely inflamed and the peritoneal cavity contained purulent fluid and feces. The child continued to vomit worms after operation and died in 60 hours; the antemortem temperature was 107.4° F (by axilla), the pulse rate 160 and the respiratory rate 48. Postmortem examination revealed a spreading peritonitis. Numerous active round worms were present in the ileum, jejunum and stomach.

Intestinal parasitism confused the diagnosis in this case but no worms were found in the appendix nor was there any evidence of their previous presence in that organ though they were present in numbers in other parts of the gastrointestinal tract. Repeated purgation undoubtedly hastened the pathologic process and contributed to the fatal result.

If parasitism were a common cause of acute appendicitis one might reasonably expect to find the disease very frequent in children in whom the incidence of parasitism is highest whereas it is actually most frequent in adolescents and young adults. One would also expect to find the association very frequent in such countries as China where intestinal infestation is widespread whereas actually acute appendicitis is almost an unknown disease in that nation (p. 48).

A few cases are on record in which an etiologic connection between parasitism and acute appendicitis perhaps existed though even in them legitimate doubts arise. Mayer reported removing the appendix in 3 of the siblings of a poor Italian family. Parasites were found in all 3 cases and in 2 instances actively motile pinworms were observed in the appendiceal lumen. He was not however prepared to say whether the finding was etiologic or merely coincidental. Frazier observed a 2½ year old Negro child with a known history of parasitism 24 hours after the onset of illness. Operation was refused by the family on the ground that the child was too young for surgery. Twenty-four hours later the boy passed a worm, was given a purgative by his mother and passed eight more. At operation performed 72 hours later a ruptured appendix was found with an ascaris free in the diffuse purulent peritoneal exudate. Clinically the child was improving when the purgative was given and it is difficult to say whether the parasites were the cause of the appendicitis or the purgative was the cause of the rupture. Cope operated on a patient with symptoms of appendicitis and found the mucosa ulcerated and the appendix crowded with threadworms. The pathologist stated that he had never before seen ulceration caused by this type of parasite. The situation was clarified when the patient continued to run a regular fever after operation and tests revealed that he had typhoid fever.

in the cecum. The farther from the cecum the examination is made, however, the more different does the bacterial flora become. Larger bacteria, especially spore-forming forms which are included in the gas gangrene group, become progressively less numerous. There is also a reduction in gram-negative organisms which usually predominate in the cecum. The closer to the tip of the appendix the material is secured, the greater the predominance of gram-positive organisms, but these also exhibit variations. Larger cocci, streptococci and diplococci disappear, as do medium-sized rods, so that finally only small gram-positive diplococci and fine gram-positive rods remain. Among the organisms which a healthy appendix may contain according to Aschoff are *B. coli* (*Escherichia coli*), gram-positive diplostreptococci, enterococci A and B, non-hemolytic streptococci, true pneumococci and on rare occasions streptococci exhibiting marked hemolysis.

Penetration of the mucosa by bacteria occurs according to Aschoff by way of single or multiple epithelial erosions at the base of a mucosal crypt. A wedge-shaped area of inflammation (leukocytic accumulation) then develops and spreads to the serous coat, advancing in the furrows of the mucous membrane, spreading from one to another, and finally involving the whole wall in a phlegmonous inflammation as the result of the confluence of various separate lesions. Spread occurs lengthwise in the muscular and serous layers, while at the same time the inflammatory process continues to destroy the mucosa if the disease has begun distally, though the spread in this layer is less rapid. The formation of intramural abscesses is characteristic.

The chief problem in the bacterial theory of acute appendicitis is to explain why bacteria which are normally innocuous are suddenly able to provoke acute inflammation. According to Aschoff, who himself granted the difficulties of finding a reasonable explanation, this is the result of stasis and stagnation which may be favored by fecaliths but which are not caused by them. The normal habitat of the diplococcus is the distal third of the organ, which explains why the pathologic process is most often located in that area.

This theory does not seem acceptable for a number of reasons: (1) Diplococci, while apparently frequent components of human fecal flora in Europeans, are not commonly found in Americans, in whom appendicitis is far more frequent. (2) Stasis does not ordinarily enhance the virulence of organisms but rather weakens it. (3) The wedge-shaped area of inflammation postulated by Aschoff has never been demonstrated by any other observer. (4) Bacteria are present in only a small proportion of early cases of acute appendicitis, and while they are frequent

The man had never had a definite attack of acute appendicitis but regarded it as quite reasonable that the shot should be in his abdomen since he was in the habit of eating numerous ducks and pheasants every year. Operation revealed an appendix weighing 98 gm and containing 38 no 6 shots all fired. The wall was greatly thinned in spots from the pressure of the shot and rupture could easily have occurred if an acute attack of the disease had taken place.

Upton (cited by Royster) reported the case of a 79 year old man who swallowed a tooth which was found 6 years later in a ruptured appendix which was in the sac of a right inguinal hernia.

Curren and Melick reported a case of rupture of the appendix treated by incision and drainage which was followed by a persistent draining sinus and a series of recurrent abscesses. When the appendix was eventually removed it was found embedded in a mass of adhesions and was so large that it presented the appearance of a constricted portion of the small bowel. In the distal end was a large round foreign body. The pathologist was unable to identify it and it was finally submitted to the department of zoology which reported it to be the vertebra of either a squirrel or a rat. The patient denied eating the first type of flesh and was not questioned about the second.

Foster and Bowers reported the case of a 22 year old soldier operated on for acute appendicitis after about 24 hours of illness following several attacks a year for the past 10 years. The appendix was found in the pelvic position and was greatly congested. The terminal portion was dilated as the result of obstruction by a sprouting seed of a piñon nut complete with hypocotyl and root. Diagnosis was complicated in this case by the fact that the patient had had his first injection for typhoid fever and his second injection for tetanus 3 hours before the onset of symptoms. The assumption was that the seed which had obviously been present for a long period of time did not cause obstruction until lymphoid obstruction which was the result of typhoid inoculation (p 62) had completed the occlusion and set into motion the chain of events that led to closed loop obstruction.

### THE BACTERIAL THEORY OF ACUTE APPENDICITIS

According to Aschoff, the chief champion of the bacterial theory of acute appendicitis whose studies on the subject began about 1908, it is possible to culture from the healthy appendix the same bacteria which can be grown from the inflamed organ. Under physiologic circumstances a healthy organ contains at the cecal end the same bacterial flora found

in the cecum. The farther from the cecum the examination is made, however, the more different does the bacterial flora become. Larger bacteria, especially sporulating forms which are included in the gas gangrene group, become progressively less numerous. There is also a reduction in gram negative organisms which usually predominate in the cecum. The closer to the tip of the appendix the material is secured, the greater the predominance of gram positive organisms, but these also exhibit variations. Larger cocci, streptococci and diplococci disappear, as do medium sized rods, so that finally only small gram positive diplococci and fine gram positive rods remain. Among the organisms which a healthy appendix may contain, according to Aschoff, are *B. coli* (*E. coli*), gram positive diplostreptococci, enterococci A and B, non hemolytic streptococci, true pneumococci, and on rare occasions streptococci exhibiting marked hemolysis.

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in advanced cases their role then is clearly not etiologic (5) Recent work by Wangenstein and his associates suggests that in a closed appendiceal loop in which bacteria are completely retained inflammatory changes do not occur until distention sets in (6) If the infectious theory of appendicitis were generally applicable it would seem that the base nearest the potential reservoir of infection in the cecum would most often be involved instead of the distal end which actually is most often and most severely affected (7) Although it is reasonable to assume that infection would explain a certain proportion of cases of disease in an organ in which a layer of lymph tissue is separated from a highly infectious lumen only by a single layer of epithelium the main point at issue remains to be explained namely why organisms present for years and innocuous for years should suddenly acquire virulence

The theory of elective localization of bacteria is even less popular now than when Rosenow first advanced it His studies dealing with acute appendicitis seem particularly unconvincing On the theory that the appendix like the tonsil filters organisms out of the blood stream he isolated organisms from the appendix and from diseased tonsils and injected them intravenously into rabbits Although many of the animals died the lesion in the appendix was never in itself sufficient to explain the death and the development of lesions in other parts of the body showed that the condition which had developed was a true septicemia Since individuals with streptococcic septicemia do not habitually develop appendiceal lesions and since in acute appendicitis a preceding focal lesion cannot usually be demonstrated Rosenow's theory seems little more than a theory and certainly without general application On the other hand acute appendicitis is probably of hematogenous origin in a small proportion of cases though the mechanism of origin remains to be clarified

It is not remarkable that pneumococci streptococci and other microorganisms which exist in the mouth and throat and are readily swallowed should be found in the appendix but whether they are responsible for acute appendicitis is another matter Even if the disease co exists with an upper respiratory infection as it frequently does the etiologic connection is dubious for one thing the strains cultured from the two areas are frequently not the same and for another similar organisms can usually be cultured from other parts of the intestinal tract in which no inflammation at all has occurred *B typhosus* and *B paratyphosus* can frequently be isolated from the appendix in acute attacks coincident with typhoid or paratyphoid fever but their responsibility for the appendiceal disease again remains to be proved

In short in the light of present knowledge while it cannot be said that no case of acute appendicitis is primarily of bacterial origin the evidence for such etiology is unconvincing and in numerous cases can be completely overthrown.

### THE OBSTRUCTIVE THEORY OF ACUTE APPENDICITIS

Van Zwalenburg in 1901 and 1905 seems to have been the first to point out that simple infection is not enough to account for the abruptness of the onset in many cases of acute appendicitis or for the severity of the tissue changes which may ensue within brief periods of time. He also suggested that the interference with the blood supply observed in many instances of the disease was better accounted for on the basis of obstruction and intraluminal changes than on an infectious basis. In a sterile organ he reasoned interference with the blood supply may do no great damage for several hours but in the appendix the situation is different and damage is likely to occur promptly because of the invasion of dead tissue by bacteria from the lumen. Van Zwalenburg also noted that Pozzi who credited the original observation to Dieulafoy had emphasized as early as 1897 that a closed loop in the appendix behaves exactly as a closed loop in any other part of the intestine and had stressed the mechanical influence of the valve of Gerlach (p. 26).

No great attention seems to have been paid to these ideas until 1914 when Wilkie writing from the clinical point of view described a type of acute appendicitis of obstructive origin and declared that it warranted far more emphasis and far prompter treatment than the infectious variety. His views attracted little more attention than had Van Zwalenburg's and it was not until 1936 that Wingensteen Bowers and their associates undertook the carefully controlled experimental and clinical studies which have made clear that the theory of obstruction is applicable in far more cases of acute appendicitis than any other theory so far advanced. These studies chiefly concern the effects of complete and incomplete obstruction the maintenance of increased intraluminal pressure isolation of the appendix as a closed loop the role of bacteria in obstructive appendicitis and interference with the appendiceal blood supply. For a complete understanding of these important studies the original publications should be read in full but the important data and conclusions may be summarized about as follows.

In the chimpanzee and the rabbit but not constantly in other species obstruction of the lumen of the appendix by ligation which spares the blood supply produces appendicitis. Elevated intraluminal pressure



causes the mucosa to secrete fluid. In the rabbit increased pressure causes occlusion of some of the vessels of the wall and the areas of ischemic necrosis thus produced introduce two possibilities. They offer a fertile field for invasion by bacteria within the appendiceal lumen and they encourage perforation at the point of weakness.

The human appendix with a normal mucosa has a secretory function. When obstruction of any kind occurs therefore the degree of the intraluminal pressure may approach that of the systolic blood pressure. If the mucosa is atrophic fluid secretion does not occur. When the pressure in the obstructed organ rises above a certain level the viability of the wall is threatened and gangrene will ensue even in the initial absence of infection. Furthermore a considerable degree of pressure can be built up even in the absence of definite organic obstruction to the lumen; this can be demonstrated by observing the resistance to the free flow of water introduced through a needle in the tip. Diseased appendices rupture at a very much lower pressure than normal appendices and experimental rupture can be hastened by the oral administration of croton oil or the intravenous injection of hypertonic solutions of sodium chloride. The minimal histologic changes found in organs removed early in the course of an acute attack of appendicitis suggest that bacterial invasion is a late phenomenon and is not an etiologic factor in acute obstructive appendicitis.

All the studies conducted by Wangenstein and his associates indicate (1) that obstruction with resultant increased intraluminal pressure is the most important factor in the genesis of acute appendicitis, (2) that infection is not primary but is a secondary result of obstruction and (3) that the course of events is on the whole what would be expected in obstruction of a closed loop anywhere else in the intestinal tract. In general it is about as follows: Blood is pumped into the terminal arteries of the appendix under the force of the systolic blood pressure. Its return through the veins of the appendix is precluded by the increase in intraluminal tension which is the result of some form of obstruction. The maintenance of this pressure-tension disturbance results in a struggle for hydraulic equilibrium. As a next step venules rupture and hemorrhage occurs into the wall. Tissue oxygenation is impaired. Finally the mucosa loses its ability to repel invasion of bacteria which enter from the lumen and begin to play a part in the pathologic process.

**Causes of Appendiceal Obstruction.** Obstruction of the lumen of the appendix may occur from a variety of causes of which fecaliths are the most frequent. They are not however necessarily a source of trouble since they are found at autopsy in large numbers of persons with no

history of appendiceal disease. The theory that they are absent in a first attack and more often present in patients with previous attacks has not been established.

The incidence of fecaliths varies widely. Fitz found them in 47 per cent of his cases and Murphy in 38 per cent of his. Bowers reported them present in 38 per cent of his simple acute cases, in 65 per cent of the gangrenous cases, in 86 per cent of the cases of generalized peritonitis and in 90 per cent of the cases of localized peritonitis, as compared with an incidence of 29 per cent in interval appendectomy and of 25 per cent in incidental appendectomy performed in the course of gynecologic operations. Tennison and Dixon reported an incidence of less than 15 per cent in purulent subacute and chronic appendicitis and an incidence of 24.4 per cent in gangrenous appendicitis. Only 0 per cent of the appendicea not containing fecaliths were gangrenous against 16.5 per cent of the appendicea which did contain them. These authors thought it not unreasonable to assume that some cases of appendicitis which might otherwise remain subacute or chronic may progress to gangrene if fecaliths are present though they will not necessarily remain uncomplicated in their absence.

Fecaliths are apparently formed from fecal material plus retained mucus which gradually becomes inspissated and incorporated with masses of dried bacteria and sometimes with other substances such as calcium salts. A central nidus such as a mass of cellulose or occasionally a foreign body is usually present and laminations due to concentric deposits surround it. Fecaliths may be hard, white and odorless or soft and brown with the odor of feces. They may be multiple.

The confusion between appendiceal calculi and appendiceal fecaliths is chiefly due to carelessness in nomenclature. The term fecalith should be reserved for noncalcified inspissated fecal masses and the term calculus for true stones which contain inorganic material chiefly calcium phosphates as well as fecal matter. The nucleus of the calculus is occasionally a foreign body or even a gallstone but more often is inspissated fecal material.

Appendiceal calculi are by no means as frequent as fecaliths. Felson and Bernhard, who reviewed the literature in connection with their report of 10 personally observed cases, found that 100 cases had been recorded up to 1947. In 7 of their own cases and in 11 of the collected cases the diagnosis had been made before operation by roentgenologic methods.

Calculi are apparently of more serious significance than fecaliths. Perforation of the appendix was associated with their presence in about

50 per cent of the recorded cases and seems inclined to occur early it was observed within 8 hours of the onset of symptoms in one of Felson and Bernhardt's cases. The conclusions of these authors therefore seem justified (1) that the diagnosis of appendiceal stone should be entertained whenever laminated calcification in the right lower quadrant is revealed by roentgenogram (2) that immediate steps should be taken to confirm or disprove the presence of the stone in the appendix and (3) that the presence of stones in the appendix is an indication for prompt appendectomy whether or not the patient has had symptoms referable to the appendix since acute appendicitis is always a possibility and since the disease in such cases is likely to run a serious course. There is never of course any reason for giving a patient with acute appendicitis barium by any route.

Calculi in the appendix occasionally reach a large size as in the case recorded by Bunch and Adcock.

A 61 year old man with symptoms suggestive of carcinoma of the cecum presented a large mass in the right lower quadrant of the abdomen. At operation this mass proved to be an enormously enlarged appendix which weighed 13.5 gm. In the distal end was a giant calculus surrounded by 4 smaller calculi apparently fragments of the parent stone which originally must have extended the length of the lumen. The appendix was not inflamed but the wall about the concretion was thinned out, the muscular layer was absent and the mucosa except in this area was hypertrophied.

The incidental role of most foreign bodies other than fecaliths and calculi in the causation of acute appendicitis has already been mentioned. Lennon in reporting a case of gangrenous appendicitis in which three impacted gallstones were present noted that only 3 similar cases had been reported up to 1936. His patient had taken a purgative.

Obstruction may be produced in addition to foreign bodies by a variety of other causes such as kinking, torsion, congenital bands and adhesions and excessive motility permitted by an abnormally long cecal mesentery (fig 16 p 24). Gray and Heifetz who were impressed by the unusual richness of the lymphoid tissue which was the prominent histologic feature in a group of mildly acute and nonacute appendices examined by them advanced the idea that either focal or diffuse hyperplasia of this type of tissue may cause sufficient obstruction in an organ with a narrow lumen to produce the symptoms of obstructive appendicitis.

In this connection Bowers and Shupe in 1942 observed in an Army camp that immediately after each new increment of troops was inoculated against typhoid fever an influx of patients with symptoms of acute

appendicitis was received in the hospital. Symptoms usually subsided spontaneously but 8 patients came to operation. The appendices were studied carefully because of the striking time relation observed in so many similar cases and the conclusion was reached that in these instances the acute disease had developed on the basis of obstruction of the appendiceal lumen by swelling of the lymphoid tissues as part of the reaction to typhoid inoculation. The writers were of course careful to emphasize that the value of typhoid inoculation far outweighs the danger of the development of acute appendicitis as a possible result of the lymphoid reaction.

A few cases have been reported in which obstructive appendicitis was caused by neoplasms. Hamilton reported a case of rupture of the appendix in which the obstructing agent was a carcinoid tumor and Howard a case in which it was a columnar cell carcinoma. In the latter case a supposed appendiceal abscess was treated conservatively and when operation was performed three months later the unsuspected malignant disease was found. Howard reasoned that the original attack of acute appendicitis was probably caused by neoplastic obstruction, the presence of which prevented complete resolution. Roberts reported a case in which a patient with the syndrome of acute appendicitis and without previous signs or symptoms of other intra abdominal disease died of a heart attack before operation could be performed. Autopsy revealed carcinoma of the rectosigmoid, secondary growths had invaded the appendix and an intracecal mass at the orifice was responsible for obstruction of the lumen.

A case of neoplastic obstruction of the appendix is included in the Chantry Hospital series.

**Case 3.** A colored woman 56 years of age had had generalized distention of the abdomen without much pain for the past year. Three months before admission to the hospital she had bled from the vagina for 3 days. A hysterectomy had been performed 14 years before. The temperature was 98.6° F, the pulse 80 and the respiration 20 per minute. Physical examination revealed abdominal distention and a possible soft mass in the right lower quadrant. Rectal examination was negative but vaginal examination revealed a somewhat tender mass in the right adnexal region.

There were 2,300,000 red blood cells and 22,800 white blood cells per cu mm with 90 per cent polymorphonuclear leukocytes. The urea of the blood was 19.5 mg per cent. Roentgenologic examination of the abdomen by flat plate was negative.

The admission diagnosis was right ovarian tumor. On the sixth day of hospitalization the patient developed a pneumonitis for which she was treated

with neoprontosil Seven days later she complained of generalized abdominal pain There were no areas of tenderness or rigidity The temperature was 100.6 F Thereafter the course was progressively downhill and death occurred on the sixteenth day of hospitalization Postmortem examination revealed generalized peritonitis due to rupture of the appendix which was the site of a carcinoid (argentaffine cell) tumor

This case is an illustration not only of obstruction and rupture of the appendix due to occlusion by a carcinoid tumor but also of the mild course of even obstructive appendicitis in middle and late life at no time were there symptoms or findings referable to the appendix

In connection with obstructive appendicitis attention should be called to the cases familiar to every surgeon of experience in which at operation as a kink is released or an organ twisted on itself is untwisted gas can be heard to escape and the appendix immediately decreases in size Such an appendix is definitely obstructed and definitely the site of a pathologic process though if it is removed early enough in the course of an attack the laboratory is likely to report only minimal tissue changes if any at all

#### MISCELLANEOUS CAUSES

The theory that acute appendicitis is more common among well to do than among poor and institutionalized subjects is not borne out by any available statistics No time need be spent either in discussing such theories as vasospasm disturbances of the autonomic nervous balance or spasm of the appendiceal musculature as the result of neuromuscular incoordination All no doubt are applicable in the occasional case but none obviously is generally applicable

How far afield theories of the origin of acute appendicitis may lead some observers is typified by the discussion which raged on the subject in the correspondence columns of the *British Medical Journal* in the late summer and fall of 1940 following a scholarly discussion of etiologic theories in the editorial columns shortly before One correspondent postulated that the cause must go far back in the patient's history otherwise the enormous amount of work done on the subject could not fail to have discovered the cause He listed as possibilities excess of fat or of carbohydrates or intolerance to them or lack of cystine in the first artificial feedings or lack of vitamin A with a history of abnormal muscular effort before the acute attack is likely precipitating causes A second correspondent was of the opinion that when a complete reaction occurs and children are no longer subjected to the ravages of hard curds in the

cradle appendicitis will be as rare as it was a generation ago. A third correspondent attributed the disease to meat eating and suggested a most unfortunate treatment consisting of an enema a week's fast nothing by mouth except sips of water and not more than half a pint a day of fruit juices and vegetable tissue together with rest and a dessert spoonful three times a day of castor oil and olive oil in equal parts. With his statement that none of his patients under this treatment had ever needed operation a fourth correspondent agreed on the ground that they would all be dead under such a method of management. One of the earlier correspondents then returned to the discussion with the statement that millions of mothers have averted attacks by castor oil in timely doses, concluding: Perhaps once in a blue moon it might precipitate rupture by an hour or so when it is imminent but how often is it imminent before the nature of the stomache is recognizable? Seldom if ever. The fact that such a discussion could take place in the columns of a highly respected medical journal in the year 1910 is as good an indication as any of the still unsettled etiology of acute appendicitis.

Note Trauma as an etiologic factor in acute appendicitis is discussed under a separate heading (p. 354) not because of its frequency but because of its possible medico-legal implications. The relationship of diverticuli of the appendix (p. 107) and of hernia (p. 160) to acute appendicitis is also discussed elsewhere.

## CLINICAL CONSIDERATIONS

From the foregoing discussion it is evident that although most of the causes listed may act as etiologic or predisposing factors in *some* cases of acute appendicitis none of them is applicable to *all* cases. As a matter of fact in numerous instances no etiologic factor at all is evident. In more than a third of the 3400 cases studied by Collins from this aspect there was no demonstrable cause. In another third the possible causes were parasites (0.79 per cent), foreign bodies (1.6 per cent) enterogenous causes (10.45 per cent) and hypertrophy of the lymphoid follicles and stenosis of and kinks in the lumen (20.6 per cent). The remaining cases were due to obstruction. The most significant consideration in this study is not the proportion or distribution of etiologic factors but the fact that obstruction of the lumen of the appendix was present in more than 81 per cent of the gangrenous cases and in almost 80 per cent of the perforative cases. Similar observations by other observers make clear the importance of the obstructive factor in both the morbidity and the mortality of acute appendicitis.

From one standpoint the etiology of acute appendicitis is at this time an academic consideration since in the light of present knowledge nothing can be done to prevent the disease. On the other hand a comprehension of the mechanism of obstructive appendicitis is a most important clinical consideration. When once its importance is realized there is likely to be prompt surgical interference in such cases and a corresponding reduction in mortality.

In the same way a realization of the possible confusion in diagnosis which can be caused by such indeterminate etiologic factors as dietary indiscretions, intestinal parasitism and upper respiratory infections which are more likely to be coincidental than causative would again lead to prompt exploration in doubtful cases. The realization that the coincidence is possible is what really matters. Unnecessary operations done under such circumstances are always much less to be regretted than are the disasters likely to ensue if necessary operations are deferred while the surgeon follows etiologic will of the wisps.

<sup>1</sup> Protein hydrolysates (Royal Society of Medicine) *Lancet* 1:723-4 (9 June) 1945.

## ( IV )

### *The Bacteriology of Acute Appendicitis*

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#### GENERAL CONSIDERATIONS

The contents of the appendix even in its normal state are essentially contaminated because the appendix is an offshoot of the cecum which of all areas of the intestinal tract is the most favorable for the multiplication of micro organisms. Under normal circumstances the micro organisms which inhabit the appendix are apparently not pathogenic. They increase in number and virulence and become invasive under two possible conditions (1) When injury to the mucous membrane occurs from extrinsic or intrinsic causes (2) When injury to the blood supply of the appendix produces damage to the wall and thus opens the door for invasion. As the pathologic process advances other bacteria, which are true secondary invaders begin to take part in it.

#### SPECIES OF BACTERIA

It was formerly believed that *Bacillus coli* chiefly in the form of *B. coli communis* less often in the form of *B. coli communior* is the chief causative organism in acute appendicitis. Although results obtained by various workers frequently differ the colon bacillus (now known as *Escherichia coli*) still seems the most frequently observed organism if all studies are considered in the aggregate.

Numerous other organisms however have been found to play major or minor parts in the process including *Streptococcus hemolyticus non hemolyticus* and *viridans*, *Staphylococcus albus* and *aureus*, *Proteus vulgaris*, *Bacillus pyocyaneus*, *Bacillus typhosus* and *paratyphosus*, Vincent's organism, pneumococci of various strains and diphtheroids. Generally speaking a thick foul purulent exudate is likely to be associated with infection by *Bacillus coli* and a thin odorless slightly turbid fluid with infection by the streptococcus.

**Gas Forming Micro Organisms** In an extensive review of the literature concerning the bacteriology of peritonitis made in 1931 but still of great value Meleney and his associates pointed out that observations



prior to 1896 belong to the aerobic era of bacteriologic knowledge. In that year Welch and Flexner reported the finding of a new micro organism, *Bacillus aerogenes capsulatus*, in 7 cases. Two years later Veillon and Zuber found anaerobes twice in pure culture, in the exudate of 21 out of 22 cases of peritonitis. Not unnaturally it was concluded that these organisms play the principal role in the production of appendicitis; that their gangrene-producing properties lead to frequent and early perforation; and that their toxins are often responsible for the fatal outcome.

Since that time opinions with regard to the frequency and importance of anaerobic organisms in appendicitis have varied considerably. Meleney and his associates in their personal study found *Clostridium welchii* in only 12 of 30 cases of perforated appendicitis, in every one of which *B. coli* was also found; in only 2 of 11 fatal cases of perforated appendicitis; and in none of 9 cases of gangrene without perforation. Their own conclusions were that no evidence exists to justify such conclusions as were drawn by Veillon and Zuber that *Cl. welchii* is only one of many organisms likely to be present in appendiceal peritonitis; and that its presence does not materially increase either the severity of the disease or the chance of a fatal outcome.

Other investigators have reported differing results. Jennings in a small but unselected series also studied in 1931 found *Cl. welchii* present in the contents of the lumen of the appendix in 90 per cent and in the appendiceal wall in localized pus collections and in free peritoneal fluid in 75 per cent of cases; blood cultures were almost uniformly negative. Altemeier in a study of 100 cases of perforative appendicitis and peritonitis in 1938 identified 16 species of aerobic and 18 species of anaerobic micro organisms. *Bacillus coli* was found in 79 and streptococcus in 44 cases. Anaerobic organisms were found in 96 cases, in only 18.5 per cent of which *Cl. welchii* was identified. *B. melanogenicum*, an organism not usually reported, was cultured from 92.7 per cent of this group of cases.

Bower, Burns and Mengle in a cultural study of 55 cases of gangrenous appendicitis with spreading peritonitis, also made in 1938, found 31 specimens positive for aerobes and 35 positive for anaerobes. Three quarters of all cultures revealed multiple organisms, the highest number in any single case being 6. *Clostridium welchii* and *B. coli* were found in association in more than half of the cases. On the basis of their findings in this and other studies these investigators concluded that *Cl. welchii* has a definite etiologic activity in the toxemia of spreading peritonitis and that its toxins are largely responsible for the lethality of the disease.

The present composite opinion concerning gas forming bacilli is that they are neither as frequent nor as important in acute appendicitis as they were once supposed to be or as occasional observers still believe them to be. On the other hand they are frequent secondary invaders and when once they appear they may do a great deal of damage because of their ability to produce toxins and because of their symbiotic effect on nonaerobic organisms.

*Interpretation of Bacteriologic Results* There are a number of possible explanations for the confusion which exists concerning the bacteriology of acute appendicitis. The most obvious is the failure to interpret results in relation to the time element in the disease. It stands to reason that the results of a culture in a case of simple acute appendicitis are likely to differ materially from the results of a culture in a case of gangrenous appendicitis and that both may differ from the results of a culture in a case of perforative appendicitis; that the results of a culture made at operation performed 6 hours after the onset of illness when the process is still confined to the appendix are likely to be at wide variance with the results of a culture made 72 or 96 hours after the onset when secondary invaders have entered the field and that the results of a culture made at operation are also likely to be at considerable variance with the results of a culture made on material secured from a drainage tube 3 or 4 days after operation when a variety of intrinsic and extrinsic conditions may have completely altered the bacteriologic picture. A mere report of results therefore without mention of qualifying circumstances can cause only confusion just as can comparisons of bacteriologic results in series of cases which are not fundamentally alike.

Techniques of examination may also be responsible for considerable differences in bacteriologic results. If cultures are not made by anaerobic methods or if the methods employed are technically inadequate anaerobic bacilli will naturally not be identified. They are not particularly easy to culture and an inexperienced or uninterested technician will always have a smaller percentage of positive results than a competent and interested one. The actual technical methods employed, the area from which the material for culture was secured, the number of examinations made and the length of time that elapsed between the taking of the specimen and the preparation of the culture must all be taken into consideration before valid conclusions can be drawn.

Bowers' studies make clear the importance of the time element. In reviewing the literature he noted that some workers had the habit of deferring cultural preparations until morning on specimens secured at night emergency operations. In an endeavor to prove or disprove his

own theory that the time element might explain some of the differences in reported bacteriologic results he made comparative studies from the same appendicectomies at various intervals after their removal. The outcome of the investigation proved the soundness of his surmise. Cultures taken immediately after or during the first hours after appendectomy showed either no growth at all or only a few colonies of bacteria in the submucosa. No great increase in the number was observed for the next 4 hours. Thereafter there was a successively heavier growth until at 16 hours there were many colonies in all layers of the appendix and at 24 hours there was a still larger number. Contrary to the usual experience Bowers had a slightly higher percentage of positive results with Gram's tissue stain than with cultural methods.

### PROGNOSIS IN RELATION TO BACTERIAL FINDINGS

Wilkie in 1912 seems to have been the first to suggest the advisability of microscopic examination in the operating room of smears from the peritoneal fluid in ruptured appendicitis to determine the necessity for drainage as well as for prognostic purposes. In his opinion drainage was unnecessary and recovery was assured if polymorphonuclear leukocytes in the fluid showed active phagocytosis particularly if the cells were ingested by macrophages. Caislaw in 1915 generally confirmed these ideas though he considered the phagocytosis of bacteria more important than the phagocytosis of cells. He also recommended that the findings be interpreted in relation to the duration of the disease. If operation were done early absence of polymorphonuclear leukocytes might be expected whereas if it were done late their absence made the prognosis poor. Degeneration of the cells was to be expected in advanced disease but in early acute appendicitis it was a bad sign.

Later investigations have in general borne out these observations. Collier and Brinkman found the prognosis grave when a smear from the peritoneal cavity showed a large number of bacteria and a small number of leukocytes and good when the findings were reversed. Meleney and his associates found that every patient recovered when smears made at operation yielded no growth or when fewer species appeared on the culture than were observed on the smear. When more varieties of organisms were found on culture than were seen on the smear more than 20 per cent of the patients died and when all forms seen on the smear grew out on the culture more than 25 per cent died. The prognosis was always worse when more than one organism was present. These observations are in agreement with those of Altemeier who ob-

served no fatalities when only one micro organism was present and in the most severe cases identified five or more

Bowers studying the problem from another angle found a direct correlation between the severity of the disease and the presence of bacteria in the tissues. Cultural methods revealed no bacteria in the appendiceal tissue in organs removed at interval appendectomy but cultures were positive in 20 per cent of acute cases and 60 per cent of gangrenous cases. A higher proportion of positive findings was obtained in acute cases with an obstructive etiology. The same correlation of bacteriologic positivity and an obstructive etiology was observed in experimental studies and the results were interpreted to mean that bacteria are forced into the tissues by increased intraluminal pressure.

Melney and his associates very sensibly noted that it is more rational from every point of view to study the material present in the peritoneal cavity and presumably left behind after appendectomy than to examine material in an organ which has been removed and is no longer a factor in the illness. Duvalmier's studies were made from this point of view. In the catarrhal type of acute appendicitis he found pathogenic organisms present only occasionally. In the phlegmonous type the exudate frequently contained streptococcus enterococcus *B. coli* and *B. perfringens*. When thrombosis of the appendicular and mesenteric vessels was present *B. coli* and streptococcus were present in most cases.

Steinberg who has studied intensively the stages of peritonitis in relation to the natural defense mechanism has arrived at the following conclusions:

When the appendiceal disease reaches the serosal coat of the appendix by gradual progression from the mucosal coat the bacteria on the peritoneal lining also multiply gradually because time is needed for them to become accustomed to their new habitat. At this stage the peritoneum is only slightly inflamed. Polymorphonuclear leukocytes which constitute the most important factor in the local struggle migrate into the peritoneal cavity and phagocytose the invading bacteria while such bacteria as pass out of the cavity through the lymphatics and capillaries are taken care of in a patient with normal reactive powers by the bactericidal substances of the blood or are ingested by the reticulo endothelial system. At this stage therefore the infectious process can be controlled by the patient's own resistance as manifested by local phagocytosis in the peritoneal cavity, the bactericidal properties of the blood and the phagocytic properties of the reticulo endothelial system.

At the end of 18 to 24 hours the bacteria in the peritoneal cavity begin to multiply and whether or not they can be controlled thereafter

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containing ingested bacteria increases. When this prognostic method is employed two precautions are essential. (1) The investigation must be complete since a study of leukocytes alone or of bacteria alone would provide a completely misleading picture. (2) The time element must be evaluated. Since conditions within the abdomen may change from hour to hour the interpretation of the findings must be tempered by some knowledge of the course of the disease and by a liberal sprinkling of common sense.

In a recent study with Martin Steinberg advanced the opinion on experimental evidence that diagnostic and prognostic conclusions are warranted on the basis of an examination of exudate taken from any portion of the peritoneal cavity except the surface area where the bacterial invasion is more massive than anywhere else. He therefore considered his own practice of making the puncture in the mid abdomen between the umbilicus and symphysis pubis entirely justified and regarded the results as representative of the degree of infection and the state of defense of the entire peritoneal cavity. In this same article it was pointed out that Steinberg's studies on the diagnostic and prognostic value of peritoneal puncture have been confirmed by Henry and Vale and by Vaccaro, Paredes and Ilie.

### CLINICAL IMPLICATIONS

The conflicting opinions concerning and the varying results of bacteriologic studies in acute appendicitis show clearly that the findings must be interpreted in relation to the duration of the disease, the time of the examination in relation to the time at which the appendix was removed and the bacteriologic technique employed. Such studies while helpful from the prognostic standpoint should not be accepted absolutely. Variations in the outcome of the disease in apparently similar patients with apparently the same pathologic processes and with identical bacteriologic findings suggest that the factor of individual resistance to infection is a highly important consideration. Meleney and his associates advanced the opinion that the higher mortality of acute appendicitis after middle age furnishes further proof of this fact.

From the practical standpoint bacteriologic studies in acute appendicitis were formerly of no great consequence since the only treatment for the disease is prompt operation. The introduction of chemotherapy has somewhat increased their value in the complicated types of disease since the effectiveness of chemotherapeutic and antibiotic agents depends upon whether or not bacteria present in a given case are respon-

depends upon the number of available polymorphonuclear leukocytes. The greater the number the more effective is the process of phagocytosis and the better are the chances that multiplication of microorganisms will be inhibited or completely checked.

As bacteria multiply in larger and larger numbers the inflammatory process becomes more and more severe and the natural mechanism of defense less and less effective. As the inflammatory process causes more and more slowing and greater and greater stagnation of the circulation fewer and fewer bacteria leave the peritoneal cavity and fewer and fewer leukocytes enter it. Finally capillary and lymphatic thrombosis and circulatory stasis almost completely inhibit both the escape of bacteria and the migration of protective leukocytes.

At the end of still another 24 hours the bacteria have achieved sufficient numerical strength and have become sufficiently potent to elaborate toxic substances which diffuse from the peritoneal cavity and affect various viscera. Their effect on the myocardium is especially disastrous for damage to the muscular structure of the heart results in further impairment of the circulation and more pronounced circulatory stasis.

According to Steinberg's studies when the defense mechanism is adequate and/or bacterial invasion is relatively avirulent free bacteria are rare and less than 2 per cent of the leukocytes contain ingested bacteria. When the defense mechanism is feeble and/or the bacterial invasion is virulent there is an exceedingly large number of free bacteria representing more than one species. The outcome in any given case of peritonitis according to this concept of the process depends upon (1) the response of the bone marrow which accelerates the production of polymorphonuclear leukocytes (2) the bactericidal activity of the blood (3) the integrity of the cardiovascular system by means of which cellular antibodies are carried to the site of bacterial invasion (4) the efficacy of the reticulo endothelial system. Against the patient's own powers of resistance are ranged (1) the potency of the invading bacteria (2) their ability to produce toxic substances and (3) the number of species present.

The probable outcome in any given case of appendicitis with peritonitis in Steinberg's opinion can be stated within 10 minutes by the examination of smears taken from the peritoneal cavity at operation or secured by abdominal puncture. A favorable outcome is indicated by the absence of free microorganisms and the presence of 25 per cent or less of polymorphonuclear leukocytes containing ingested bacteria; if the percentage is more than 25 even if free bacteria are scanty the prognosis is increasingly less favorable as the proportion of leukocytes

## (V)

### *Morphologic Aspects of Acute Appendicitis*

BILL HART M D \*

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Present knowledge of the cause and evolution or natural history of acute appendicitis and of the complications which the disease may produce is derived from clinical observations on individual patients and amplified by observations made on appendices at operation and at necropsy and by the gross and microscopic examination of such appendices

The most advantageous use is made of specimens removed at operation if they are at once fixed in 5 per cent formalin A few hours later following external examination a longitudinal section is made through the distal third of the organ from the free margin toward the line of attachment of the meso appendix to permit examination of the wall and to expose the contents A longitudinal section of the distal third including the tip and a transverse section from the proximal third are usually preserved for microscopic study

The microscopic structure of the normal appendix has been described in detail elsewhere (p 27) A knowledge of the microscopic structure is essential for a comprehension of the pathologic process of acute appendicitis since it furnishes clues to the mechanism by which the disease develops It also yields information concerning the possible effects of the disease process on the appendix itself on the tissues in its immediate vicinity and on distant parts

#### THE PATHOGENESIS OF ACUTE APPENDICITIS

It is important to remember that each appendix removed surgically because of acute appendicitis represents only a single stage of the disease process It resembles a single exposure in a moving picture

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sive to them. A possible practical result of a belief in the importance of anaerobic bacilli, whether as primary organisms or secondary invaders, might be the use in selected cases of the prone method of drainage through an open wound, since anaerobic bacilli cannot continue to multiply in the presence of a free supply of oxygen.

Fig 26 Herniation of mucosa through the muscular coats with acute appendicitis (x 20). A 32 year-old white man complained of pain in the right lower abdomen for 2 days. At the time of admission to the hospital there was marked tenderness in the right lower abdomen with rigidity. There was some rebound tenderness. The white blood cell count was 19,950 per cu mm. Immediately following admission appendectomy was performed. The appendix was 7 cm long with the proximal end 1 cm and the distal third up to 1.7 cm in diameter. The surface was roughened and ragged and appeared injected. Microscopically an outpouching of the mucosa through the muscular coats thinned the wall. The inflammatory process was most marked about the herniation. The tissue elements of the serosa were spread apart and were densely infiltrated with neutrophilic granulocytes. A layer of fibrinopurulent exudate covered the serosal surface (S 135-44).



If the cause or causes of acute appendicitis were known the manner of inception of the process could be better understood. A constant predisposing factor is the usual presence in the lumen of the appendix of microorganisms which are of varying virulence and which are all capable of producing an acute inflammatory reaction in the organ (p 67). The presence of microorganisms alone is however obviously insufficient to cause acute inflammation of the appendix since in spite of their constant presence relatively few individuals in each decade of life of the total population develop the disease. Another initiating factor is necessary.

Appendicitis like inflammatory processes elsewhere is a reaction to an injury but the exact manner in which the injury is initiated is not known. Although the role of foreign bodies in causing the injury has been popularly accepted there are serious objections to it (p 55). Fecaliths are frequently present in normal appendices at necropsy and their relative frequency in appendices that are the site of acute in



Fig. 25 *Enterobius vermicularis* in lumen of the appendix ( $\times 20$ ). A 9 year-old white girl complained of pain in the right lower abdomen for 10 days. At the time of admission to the hospital there was some tenderness over the umbilical region. The temperature was 99.4° F. The white blood cell count was 14,200 per cu mm with 70 per cent neutrophils. On the second day following admission appendectomy was performed. The appendix, which measured 6 by 0.5 cm, disclosed no changes except that it contained *Enterobius vermicularis*. In microscopic preparations segments of *Enterobius* were seen in the lumen; while the mucosa and the other layers were intact. Subsequently ova of *Enterobius* were discovered in smears from the anal region (S 845-45).

It does not show the beginning of the reel that is the mode of origin of the process. In fact the initial phase of acute appendicitis which might throw light on the mode of its inception is not ordinarily encountered though ample observations have been made on the successive stages of the disease and on the complications which may develop. When the acute process has once started it usually proceeds through various stages in quick succession. Modified by anatomic and other circumstances it passes to the phase of detectable clinical signs and symptoms either of the disease itself or of its complications. It rarely subsides. It is therefore reasonable to describe degrees of the inflammatory process rather than types of appendicitis. From the many single exposures or still pictures of acute appendicitis a sequence may be evolved which provides the natural history of the process.

involved. When the inflammation is well established in the distal third the presence of a fecolith will interfere with the continuity of the lumen. The contraction of the uninvolved or less involved proximal portion will imprison the concretion and cause further obstruction which will in turn increase stagnation of the contents of the appendix and will further interfere with the blood supply of the overlying or adjacent distal portion of the wall. The eventual result may be focal necrosis and perforation. The fecolith thus is a source of complications rather than the cause of the inflammation. The chain of events is first inflammation and then obstruction not the reverse.

### STRUCTURAL CHANGES IN ACUTE APPENDICITIS

An appendix is removed surgically under one of three circumstances (1) It may be uninvolved itself but be removed in the course of an operation performed for other causes in the peritoneal cavity to prevent possible future involvement (2) When the patient has signs and symptoms that mimic those of acute appendicitis an exploration of the appendiceal region seems to be warranted and the surgeon finds the appendix uninvolved yet removes it (3) When an acutely inflamed appendix produces clinical signs and symptoms characteristic of the disease the diagnosis is correctly made and an appendectomy is performed.

Clinically the following stages of acute appendicitis might well be distinguished (1) The initial stage in which the process starts with prodromal clinical manifestations but with no detectable pathognomonic signs and symptoms (2) The florid stage in which the usual clinical manifestations and the signs and symptoms of acute appendicitis are present (3) The final stage in which complications are present or regression occurs.

The degree of change observed in an appendix removed in the florid stage that is following the onset of clinical manifestations depends on a number of factors including (1) the cause of the inflammatory process (2) the intensity and extent of the inflammation and (3) the presence or absence of concretions in the lumen. The degree of change also depends upon the time elapsed between the inception of the lesion and the appearance of the clinical manifestations and the time of removal of the appendix.

Corresponding broadly to the clinical stages of appendicitis just listed the following end stages of the inflammatory process may be distinguished in removed appendices (1) appendicitis acute slight

inflammatory processes is not convincing evidence that they are the initiating cause. Fecaliths are the product of stagnation of the contents of the appendix. The presence of a fecalith increases further the stagnation which predisposes to injury of the wall making it permeable to the organisms present. *Enterobius vermicularis* (p. 52) is often present in the appendix without producing an acute inflammatory lesion (fig. 25) and it may therefore be assumed that ordinarily no causal relation exists between *Enterobius* infestation and acute appendicitis.

The presence of one or more outpouchings or herniations of the mucosa toward the external layers may sometimes be a predisposing factor. Stagnation of fecal content in herniations that are false diverticula may produce an injury to the mucosa leading to so called diverticulitis and appendicitis (fig. 26). Acute appendicitis produced by this mechanism is however limited at most to 1 or 2 per cent of all cases.

Whenever the surface epithelium is injured and entrance of microorganisms into the tunica propria of the appendix is thus facilitated an inflammatory process will surely follow. A likely mode of inception of such an injury is transient or prolonged interference with the blood supply of the appendix. Even a temporary interference with the blood supply of the appendix whether arterial, venous or both may injure the mucosa sufficiently to render it permeable to infection by the content of the appendix. There are ample opportunities for a transient circulatory disturbance to occur. The normal efforts of the cecum to expel its content may so change its position as to cause a pull on the appendix twisting its mesentery and temporarily interrupting its blood supply. A mobile cecum with large excursions may similarly cause twisting of the appendiceal vessels and produce temporary vascular occlusion sufficient to injure the lining epithelium. Interference of this kind may also result when the cecum propels its content against a hindrance created by adhesions that keep the transverse colon at an acute angle to the ascending colon.

Whatever the cause when an injury to the mucosa occurs a minute ulcer develops at the site of injury and fibrin, red blood cells and neutrophilic granulocytes ooze into the lumen. A focal acute inflammation of this sort appears to be the initial stage of acute appendicitis in most instances. Once established the inflammatory process proceeds by direct extension through tissue and lymph spaces involving in succession the submucosa, the muscular coats and the serosa. Occasionally the inflammatory process involves the distal third of the appendix at once. In all instances of acute appendicitis this third is most severely



Fig 29 Appendix with appendicitis acute. The appendix was removed from an 11 year old white boy 27 hours after the onset of clinical signs and symptoms. It was 7.5 cm long, slightly curled and 1.3 cm in diameter at its mid portion. The serosal blood vessels were prominent and the surface was dull gray and red (S-490-47)



Fig 30 Microscopic preparation of appendix shown in fig 29 ( $\times 20$ ). The lumen contained a hemorrhagic fibrinopurulent exudate which was attached to the wall where the mucosa was missing. All the layers were spread apart and infiltrated with neutrophilic granulocytes. The serosa was broadened by a fibrinopurulent exudate (S-490-47)



Fig 27 Appendix with appendicitis acute The appendix of an 11 year-old white boy was removed 24 hours after the onset of clinical signs and symptoms It measured 8.5 by 1 cm the distal third was enlarged up to 1.3 cm in diameter The serosal surface was dull gray red with the blood vessels prominent (S-453-47)



Fig 28 Microscopic preparation of transverse section of appendix shown in fig 27 ( $\times 20$ ) The lumen contained a hemorrhagic fibrinopurulent exudate which was attached to the wall where the mucosa was missing All the layers were spread apart and infiltrated with neutrophilic granulocytes The serosa was broadened by a fibrinopurulent exudate (S-453-47)

Fig 33 Microscopic appearance of middle third of appendix shown in figs 31 and 32 (x 20). A longitudinal section of the appendix disclosed fairly good preservation of the surface epithelium. There was dense infiltration with lymphocytes, many plasma cells, some large mononuclear cells and eosinophilic granulocytes in the tunica propria where lymph follicles were absent. All the layers were spread apart. There was margination of the white blood cells and an organizing fibrinopurulent exudate broadened the serosa (S-531-47).

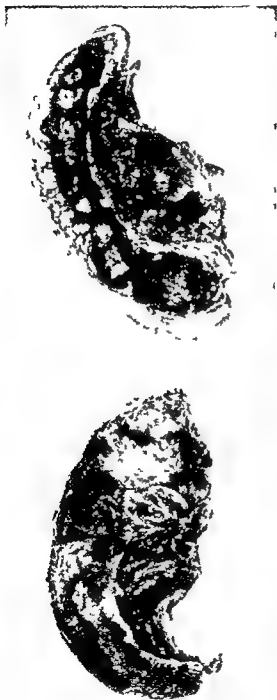


(2) appendicitis acute (3) appendicitis acute with perforation (4) appendicitis acute with gangrene (5) appendicitis subsiding (so-called chronic) and acute and (6) involution of the appendix.

**Appendicitis Acute Slight** In the initial clinical stage of acute appendicitis when the lesion is not yet capable of producing pathognomonic signs and symptoms, no gross change is observed in the excised appendix other than some dilatation of the serosal blood vessels. In a microscopic preparation of the longitudinal section from the distal third, the lumen contains a few neutrophilic granulocytes. The surface epithelium here and there is lifted off or is missing, and there are minute aggregations of neutrophilic granulocytes in the tunica propria. In the capillaries of the tunica propria, there is margination of white blood cells. No change is noted in the submucosa, muscular coats, and serosa.

**Appendicitis Acute** In this stage of the process, the appendix appears grossly swollen and reddened, and flakes of fibrin cover the surface. Usually, the distal third is most decidedly involved, and its diameter is two to five times the usual diameter of the appendix (figs





Figs 31 32 Appendix with appendicitis acute The appendix was removed from a 32 year old white man 5 days following the onset of clinical signs and symptoms At operation there was pus in the peritoneal cavity The appendix was 8.5 cm long and 2.4 cm in diameter near its proximal end it tapered to 1.5 cm at the tip Up to 2 cm of meso appendix was attached The

surface was dull red brown and covered by a thick gray film The blood vessels were injected A fecal concretion 2 by 1.4 by 1.2 cm was wedged in the lumen at the proximal end The rest of the lumen contained thick creamy liquid and 12 additional concretions measuring from 0.3 to 0.8 cm in diameter (S 531-47)

27, 29) The lumen contains some liquid feces mixed with blood and pus. Occasionally one or more fecal concretions are observed in the liquid (figs 31-3). The wall is swollen and discolored; it is gray and speckled with red and brown. The inflammatory process seems to fade gradually toward the proximal third of the appendix.

On microscopic examination a longitudinal or transverse section of the distal third of the appendix presents all the usual evidences of an acute inflammatory process. The lumen contains some fecal material mixed with some fibrin, red blood cells and many neutrophilic granulocytes. This hemorrhagic fibrinopurulent exudate is attached to the surface in areas in which the mucosa is missing (figs 28-30). All the layers are spread apart and are densely infiltrated with neutrophilic granulocytes. There is usually margination of white blood cells in all the capillaries. The serosa is broadened by a layer of fibrinopurulent exudate. The inflammatory process is of almost even distribution about the entire circumference of the distal third of the appendix and fades off gradually toward the proximal third. Occasionally, however, the entire length of the organ is evenly involved almost to its base at the cecum (figs 31-2).

The degree of inflammatory process just described gives rise to the classical clinical signs and symptoms of acute appendicitis in the florid stage. Though the inflammatory process is still localized, the gradual further deterioration of the circulation in the wall of the appendix may lead to focal or massive necrosis with perforation and then to focal or diffuse peritonitis. Inflammation that has reached this degree seldom if ever subsides and unless the appendix is removed promptly complications usually follow.

*Appendicitis Acute with Perforation.* A severe inflammatory process in the appendix with focal necrosis and perforation most frequently occurs when one or more fecal concretions are present in the lumen. These concretions interfere with the continuity of the lumen and lead to stagnation of the contents in the distal portion. Moreover, contraction of the uninvolved or less involved proximal portion of the appendix imprisons the concretion, causing complete obstruction of the lumen, stagnation of content and further interference with the blood supply of the overlying or adjacent distal portion of the wall. The result is focal necrosis and perforation at the weakest point of the wall, which is usually just behind and adjacent to the concretion, not over it (figs 34-5).

Grossly the appendix is swollen and discolored. It is widened at the site of the concretion and distal to it though proximal to the concre-



Fig 84 Appendix with appendicitis acute with perforation The appendix was removed from an 8 year old Negro boy 38 hours after the onset of clinical signs and symptoms The appendix was 6 cm long over the distal third it was enlarged to 1.5 cm in diameter The wall was discolored dark red and brown There was an area of perforation 0.2 cm in diameter 0.8 cm proximal to the tip The fecalith filled the lumen just proximal to the site of perforation (S 1762-47)

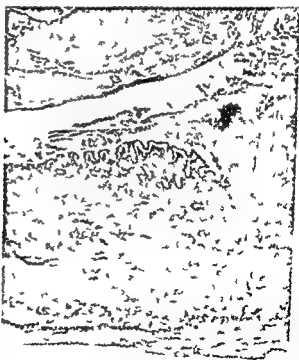


Fig 85 Microscopic preparation of appendix shown in fig 84 ( $\times 20$ ) In the lumen there was some fecal material with a laminated pattern containing lavender stained deposits All the layers were spread apart and densely infiltrated with neutrophilic granulocytes The mucosa was missing near the area of perforation and was replaced by a hemorrhagic fibrinopurulent exudate (S 1762-47)

tion it may be of the usual width. The site of the perforation is a minute opening with ragged edges surrounded by a dirty brown friable zone.

The presence about the site of perforation of fecal material and of necrotic tissue debris almost invariably results in the complications of focal or diffuse peritonitis. Depending upon the intensity of the process and the rapidity of its progress as well as on the position of the appendix a walling off by the omentum or by loops of small intestine may occur. The walling off is frequently accompanied by focal accumulation of pus in a periappendiceal abscess. A diffuse peritonitis may however result.

The clinical signs and symptoms of acute appendicitis with perforation frequently appear early and with dramatic severity and therefore give ample warning which leads when it is heeded to early removal of the damaged appendix.

*Appendicitis Acute with Gangrene.* An acutely inflamed gangrenous appendix is grossly discolored red brown or black and has the form of a friable sac containing feces and pus (figs 36-7). Microscopic examination reveals a fibrinopurulent exudate in the lumen, absence of the mucosa and either ghosts of the remaining layers or a necrotic tissue debris in their place. Small remaining islands of the wall are densely infiltrated with neutrophilic granulocytes or their cellular and nuclear fragments. Massive necrosis called gangrene occurs in part or in most of the appendix when for some reason the blood supply is suddenly and permanently cut off. The culture medium that the dead or dying tissue provides for the micro-organisms present in the lumen serves as a bridge by which they spread with great rapidity in the immediate vicinity and all over the peritoneal cavity. The fulminating process thus permits little time for hesitation in regard to surgical intervention and excision of the dead appendix.

*Appendicitis Subsiding (So Called Chronic) and Acute.* It is still possible for the inflammatory process in the appendix to subside when it is acute but slight or when it has just begun to produce clinical signs and symptoms. Subsidence occurs however only when the content of the appendix is liquid and when the lumen can be emptied. Depending upon the extent of the initial lesion the process of healing may be followed by some scarring in the wall of the appendix and by partial or complete obliteration of the lumen (p 103). An appendix removed at this stage appears grossly more firm and more rigid than a normal appendix. Microscopically the tissue elements are somewhat spread apart. There is an infiltration with eosinophilic granulocytes which are



Fig 36 Appendix with appendicitis acute with gangrene. The appendix was removed from a 25 year old Negro 72 hours following the onset of symptoms. At operation there was perforation of the appendix and about 20 cc of pus with a fecal odor was contained in the cul de sac. The appendix was 5 cm long club shaped and enlarged to 2 cm in diameter at the distal end. It was enveloped in an inflamed portion of omentum 4 by 2 by 1 cm. The appendix was discolored brown and black. About 2 cm proximal to the distal end there was an area of perforation 0.4 cm in diameter. The lumen contained a concretion and fecal material (S-457-47)



Fig 37 Microscopic appearance of the appendix shown in fig 36 ( $\times 20$ ). The lumen contained a fibrinopurulent exudate and a necrotic debris. All the layers were spread apart and lacked nuclear staining. A fibrinopurulent exudate covered the serosal surface (S-457-47)

adipose tissue and with loose or more dense fibrous connective tissue (fig. 38). The circular and longitudinal muscle layers are delicate. Occasionally groups of ganglion cells are conspicuous between these two layers. It is scarcely conceivable that an acute inflammatory process could arise in an appendix which presents such a degree of involution.



Fig. 38 Involution of the appendix ( $\times 20$ ). The appendix was removed from a 45 year old white woman in the course of an operation for uterine suspension. It measured 3 by 0.4 cm. and appeared threadlike. Microscopically the lumen was absent and fibrous connective tissue and lobules of adipose tissue filled the space surrounded by the delicate muscular coats (S 940-47).

numerous in the mucosa and submucosa but which also are frequent in the intermuscular connective tissue. Occasional scattered lymphocytes or groups of lymphocytes, plasma cells and large mononuclear cells are also seen particularly in the outer layer of the muscular coat and beneath the mesothelium of the serosa. When the lumen is obliterated connective tissue and lobules of adipose tissue fill the space. Lymph follicles are occasionally present.

An appendix removed some time after the clinical signs and symptoms of acute appendicitis have subsided (the so called interval appendix) may present the gross and microscopic appearance just described. The stage of the inflammatory process in an appendix of this kind may be called chronic subsiding. If after recession the acute process again flares up it may be referred to as appendicitis chronic and acute.

*Involution of the Appendix.* An involuted appendix has no lumen or has a lumen only near its base. It is of small caliber and may be a mere thread with diminutive layers of muscular coat. Microscopically the space usually occupied by the mucosa and submucosa is filled with

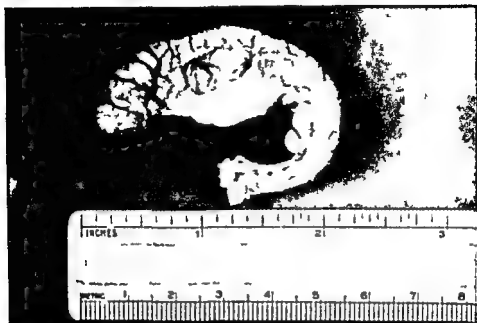


Fig. 39A Acute obstructive appendicitis. Note distention of appendix distal to point of obstruction and normal appearance proximal to it.

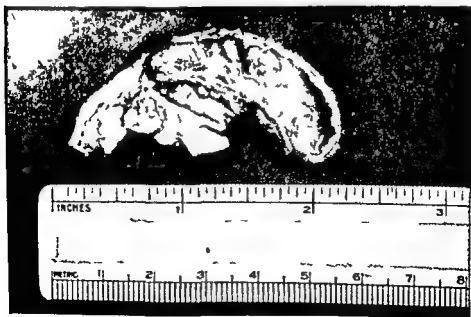


Fig. 39B Longitudinal section of appendix shown in fig. 39A. Note fecalith in lumen.



## ( VI )

### *The Pathologic Process in Acute Appendicitis*

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#### THE PATHOLOGICAL PROCESS IN OBSTRUCTIVE APPENDICITIS

Champions of the obstructive origin of most cases of acute appendicitis take the general position that the pathologic course of events is initiated by mechanical causes and that infection is a secondary and not a primary phenomenon. The arguments for this concept—which I myself hold—have been set forth in detail elsewhere (p. 59).

The following description of the pathologic process in obstructive acute appendicitis is taken chiefly from Bowers' comprehensive exposition of the subject.

In the early stages of obstructive acute appendicitis, gross inspection of the appendix is likely to reveal nothing more than increased turgidity which is the result of edema. A little later, engorgement and tortuosity of the serosal vessels are observed due to venous stasis which is the result of early distention or to hyperemia which is the result of infection. The gross appearance in other words of the obstructed and non-obstructed appendix is identical at this stage.

Later the two types may be differentiated. The obstructed appendix always becomes more or less tensely distended distal to the point of obstruction (fig. 39A, B) while proximal to this point there is a sharp transition to normal tissue. Distally the wall is thinned and is covered with a shaggy green to yellow fibrinous exudate. Proximal to the obstruction only edema and congestion are noted. The omentum or adjacent loops of small bowel or both are often adherent to the area of exudate. When the process has progressed to gangrene the obstructed appendix is tensely distended and friable. Often it is surrounded by cloudy to purulent fluid which may or may not be sterile. When perforation occurs the gangrenous distal portion of the appendix frequently sloughs off into the resulting abscess cavity and thereafter because distention has been relieved the obstructing mechanism may be difficult to demonstrate.

Because of the small caliber of the vessels of the mucosa of the appendix pressure necrosis occurs promptly as the result of distention which itself is the result of obstruction. The submucosa is thereby exposed to invasion by the bacteria normally present in the appendix and a

related to the pathologic process, the correlation is made under the appropriate heading (p 135)

### PERITONITIS

An important consideration to be borne in mind in the discussion of the pathologic process in acute appendicitis is that although the pathologist may and usually does describe peritonitis as part of the disease process the clinician must not so regard it. When once the disease process has passed outside of the appendix as it has passed when peritonitis occurs he is no longer dealing with acute appendicitis. He is dealing with a complication which it should be his chief objective to forestall. The distinction is not hair splitting or merely academic. Failure to make it is in fact one of the reasons for the confusion still apparent in the textbook descriptions of acute appendicitis in the teaching concerning it and as a final result in the therapy of the disease.

Peritonitis may develop during any stage of the disease process in acute appendicitis but is most frequently associated with rupture. Although rupture may occur at any area in the appendix it is perhaps most frequent at the tip. It does not seem to be most frequent at the exact point of blockage in obstructive appendicitis where it might reasonably be expected to occur. Sometimes there is more than one perforation. When gangrene is part of the pathologic process perforation is likely to occur earlier in the disease. Sometimes especially in obstructive appendicitis with massive gangrene what amounts to massive perforation occurs the whole gangrenous portion of the appendix sloughing off and lying free in the peritoneal cavity or within an abscess.

*Incidence* The exact incidence of appendiceal peritonitis is debatable for several reasons the first of which is the somewhat surprising one that there is no universal agreement on the meaning of the term. Some writers define peritonitis as inflammation of the peritoneal cavity following perforation of the appendix. Others more logically include in the definition any invasion of the cavity by the appendiceal process regardless of whether or not perforation has occurred. It is possible for a diffuse and sometimes a fatal peritoneal reaction to occur when the appendix though still intact is gangrenous or less often when the disease is merely suppurative. The gross contamination that accompanies perforative appendicitis may be lacking but the difference is merely one of degree. When once organisms have passed into the peritoneal cavity peritoneal contamination has occurred. It is true

heavy cellular exudate develops at this point. Neutrophilic infiltration is usually the only significant histologic change.

In the early stages of an obstructive process in the appendix the submucosa, the loose connective tissue of which lends itself to the accumulation of fluid and cells, may be the first layer to show edema and neutrophilic infiltration. Changes are first observed around the blood vessel walls, though margination of leukocytes in the vessels may also be seen as the result of vascular stasis associated with obstruction.

The muscularis tends to resist distention and as the result of its density it is late in presenting leukocytic infiltration. In the obstructive type of disease it is a strong barrier to early perforation. As it becomes thinned by distention, however, and as its muscle fibers are separated by the accumulation of inflammatory exudate, their continuity is broken and microscopic gangrene becomes evident.

According to Bowers differentiation of the obstructive from the infectious type of acute appendicitis can be made at once by examination of sections. In the obstructive type of disease thinning and distention are the rule, while the nonobstructed organ is thick-walled and soggy. Bowers also emphasizes the importance of studying the appendiceal lumen on the ground that its conformity and size, as well as its contents, clearly distinguish the obstructive from the infectious type of disease.

To prove his point about the thinning of the wall of the appendix in obstructive disease, Bowers made careful micrometric studies. In normal organs the thickness of the wall was essentially the same at the base and near the tip. In nonobstructive acute disease the wall was increased in thickness by about 25 per cent because of infiltration with fluid and leukocytes, the greatest increase occurring near the base. In obstructive disease the wall, by actual measurements, showed marked thinning distal to the point of obstruction, while if gangrene had occurred the thickness of the wall in the distal portion of the appendix was only about a third that in the proximal portion.

Changes in the size of the lumen could be readily correlated with changes in the thickness of the wall. In normal appendices the lumen was moderately bulbous, the diameter at the tip being about three times the diameter of the base. In nonobstructive acute appendicitis the lumen showed a moderate uniform increase in diameter, usually because of the accumulation of pus. In obstructive acute disease the diameter of the lumen frequently was more than 10 times the average diameter of the tip of a normal appendix.

The clinical aspects of obstructive acute appendicitis are very clearly

appendix and is essential to the defense mechanism rather than inimical to it. Their reasoning is that the diffuse involvement provides an extensive surface from which bacteria are removed from the peritoneal cavity, that great numbers of leukocytes migrate to the peritoneum and that phagocytosis is more effective since the bacteria are distributed over a wide area and the leukocytes are confronted by relatively few microorganisms. In line with this concept they suggest that the current terms of diffuse and spreading peritonitis be replaced by the terms regressing and progressing peritonitis to indicate respectively recovery and an adverse outcome.

Statistical data on peritonitis are stated elsewhere (p. 192) but it might be mentioned here that Bower has estimated that in every 100 cases of acute appendicitis the serous coat of the appendix is intact in 66 and ruptured in 34; that in the 34 ruptured cases the peritoneal involvement is of the spreading type in 14 and of the abscess type in 12 and that the remaining 8 cases are in process of localization and will so remain unless they are converted by the surgeon into instances of spreading peritonitis. Incidentally Bower has also estimated that 1 of every 200 intact appendices is ruptured by the surgeon at operation.

*Pathologic Process.* When peritonitis of any degree has developed the peritoneal coat of the appendix is thickened and edematous and the capillaries are engorged. The free border of the greatly thickened mesentery is usually adherent to the cecum or ileum. The omentum is greatly thickened as the result of edema, increased vascularity and neutrophilic infiltration. The preperitoneal tissues are usually somewhat edematous and the parietal peritoneum is injected. The degree of hyperemia of the visceral peritoneum as well as the presence or absence of adhesions depends upon the time lapse between perforation and operation and upon the patient's own resistance. The most usual pathologic changes in the mesentery are congestion and infiltration; in extreme cases thrombosis of the mesenteric veins may be observed. In the obstructive type of disease the mesenteric process is likely to be vascular alone. In the infectious type the thrombi are infected and extension of the process may take the form of portal thrombosis (pyelephlebitis) and hepatic abscess (p. 224).

Dvuzhina, who studied extra-appendiceal pathologic-anatomic changes in 100 clinical and 2 cadaveric cases of acute appendicitis, found the mesentery involved in every instance regardless of type. The changes in the omentum were dependent upon the intensity of the intra-appendiceal inflammatory process. Gross changes were found in the ileocecal lymph nodes in only 8 of the 102 cases but on microscopic

that the first stages of the peritoneal reaction are protective but that does not alter the fact that the peritoneum has become involved that the appendiceal disease has ceased to be intrinsic and that dangerous and even fatal consequences may ensue

A second reason for uncertainty concerning the exact incidence of appendiceal peritonitis is confusion in the usages of the terms spreading (generalized diffuse) and localized peritonitis. Some observers object to the term spreading on the ground that there is a natural tendency toward localization in all cases even when the disease process is no longer confined to the appendiceal region. There is also objection with considerable reason to the terms localized and generalized on the ground that no surgeon no matter how large his incision can determine the extent of the peritoneal process. It is true that generalized peritonitis if the term is used in its most literal sense can be only an autopsy diagnosis. Bower has discussed the matter very well. When the appendix perforates in a patient whose reactive capacity is normal the gross changes that follow can be divided for all practical purposes into three zones. In the central or inner zone is part or all of the perforated appendix partly or completely covered by omentum or by fibrinous or fibrinopurulent exudate. In the middle zone are the cecum adjacent loops of ileum and the omentum all adherent to each other and partly covered with exudate. Both of these zones are clearly visible to the surgeon at operation. The outer or peripheral zone however is not visible and should not be seen. As a result the changes in it are and should remain indeterminate. In other words it is not possible for the surgeon to determine at operation the exact extent of peritoneal involvement unless as Ladd indignantly expressed it he performs a very improper operation.

All of this is quite true. On the other hand the distinction between localized and generalized peritonitis is clear to all surgeons of experience who without transgressing the bounds of surgical propriety frequently have sound reasons for stating that one variety or the other is present in any given case. The nomenclature is also useful in emphasizing the relative gravity of the two conditions localized peritonitis implying that the process is still confined to the region of the appendix and generalized peritonitis implying that it has spread to other parts of the peritoneum though how far it has been emphasized it is not the business of the surgeon to determine.

In this connection there should be cited the theory advanced by Steinberg and Martin on the basis of animal experiments that a diffuse peritoneal involvement takes place shortly after perforation of the

occurred as delivery of the appendix was almost complete so that only the peritoneum adjacent to the incision was contaminated most of the patients in the Pennsylvania series lived though as several similar cases in the Charity Hospital series prove protection is not always complete even under these circumstances

The chief cause of the accident in most cases of this kind is an abnormal location of the appendix plus the practice of out of sight manipulations Whatever the reason the peritoneum is unprepared to handle the rupture there is no pre formed peritoneal exudate and to quote Bower again every movement of the surgeons hand is attended by trauma to or death of endothelial cells with consequent exposure of lymph and arterial capillaries This is precisely what happens in rapidly advancing acute appendicitis when perforation occurs into an unprepared peritoneal cavity

When the natural defensive mechanism of the peritoneum can be called into play as it can be when the disease process is gradual the first result is the production of an exudate rich in leukocytes and fibrin The presumed protective action of these leukocytes is described elsewhere (p 71) The fibrin causes an agglutination of serous surfaces by means of which contaminated portions of peritoneum are separated from uncontaminated portions and there is a mechanical localization of the infectious process In the course of localization loops of small intestine become agglutinated to the appendix and cecum The great omentum for reasons not yet understood migrates to the site of the rupture and the formation of adhesions begins Intestinal paralysis (ileus) by limiting movement in the affected area takes part in the defensive mechanism

The characteristics of the fluid in the peritoneal cavity depend upon the state of the appendix and the time at which the study is made as well as upon the bacterial flora (p 69) Soon after perforation the fluid is thin odorless clear or slightly cloudy and moderate in amount If localization occurs its characteristics are likely to remain unchanged If the process continues to spread the fluid increases in amount and becomes progressively more turbid Eventually it is seropurulent or purulent and malodorous If the appendix is gangrenous and the disease process is extensive the fluid is likely to be bloody The exudate tends to collect in dependent pockets such as the pelvis the lumbar gutter especially on the right and the intestinal flexures The multiple abscesses which sometimes form must be distinguished from primary abscess formation in the appendix itself

examination diffuse hyperplasia dilatation of the lymph vessels and accumulation of lymphocytes were found in many other cases In phlegmonous and perforative appendicitis there were microscopic changes in the peritoneum as well as in the muscle fibers though in catarrhal appendicitis no changes were observed in the musculature

If the serous coat is inflamed but the appendix is not ruptured protective adhesions may develop sometimes in the form of dense cicatricial bands more often they are filmy and cobweb like They may involve large areas or may be limited to patchy areas on adjacent structures When rupture occurs the omentum and intestines may be found matted together about the appendix sometimes completely sealing over the area of perforation If perforation has occurred into the mesentery the appendix will be completely hidden by omentum ileum or cecum Occasionally instead of rupturing into the peritoneal cavity the appendix itself (or the abscess that has formed from a previous rupture with localization) may drain into some adjacent organ such as the bladder or some part of the intestine (p 236)

The progress of peritoneal inflammation depends upon a number of factors the most important of which is the rate at which the primary appendiceal process proceeds When progress is gradual as is usual in the infectious type of disease peritoneal soiling is gradual and the defense mechanism has time to become operative When rupture occurs abruptly as it so often does in the obstructive type of disease the appendiceal contents which are always contaminated and which may under these circumstances be virulently infectious are spilled into a cavity whose protective defenses have not yet been mobilized

Bower has pointed out that rupture into the unprepared peritoneal cavity is comparable to rupture at operation of the intact appendix in which inflammation has not yet reached the peritoneal coat This accident occurred 70 times in 19938 cases of acute appendicitis included in the first report of the Pennsylvania Commission on Acute Appendicitis Mortality and 58 of the patients died (82.85 per cent) In the absence of this accident probably none of this group would have died The duration of illness among these patients averaged only 48 hours their average age was only 21 years the average temperature was only 100° F and the average pulse rate was only 104 Many of the patients developed hyperpyrexia and tachycardia immediately after operation and some of them died so promptly that the abdominal wall was rigid at death and distention had not had time to develop Some died in toxic delirium never regaining consciousness When rupture

dogs led them to certain conclusions which had not hitherto been advanced concerning the mortality. In substance their experiments showed the following points:

1. Animals that best maintained the normality of the prothrombin time throughout the postoperative period tended to survive the crisis of their peritonitis. When the prothrombin time was not within the normal range death was likely to occur.

2. Evidence of spontaneous lysis of plasma fibrinogen and fibrin occurred with much greater frequency in animals that died than in animals that survived. Lysis invariably occurred in samples drawn within the 12 hours preceding death.

3. Sharp fluctuations in available fibrinogen nitrogen were observed throughout the course of the disease in all animals. Levels approximately 65 per cent greater than the pre-operative values were observed in the survivors. Correlation between the fibrinogen nitrogen levels and the activity of fibrinolytic factors could not be clearly established.

4. Animals that possessed a sufficiently high titer of the anti-fibrinolytic factor in the blood seemed best prepared to withstand the deleterious effects of appendiceal gangrene and peritonitis.

■ The pre-operative administration of trypsin in amounts sufficient to build up antiproteolytic activity of the serum had a significant influence on survival. Only 1 of the 15 animals thus treated died while the mortality in the 91 animals used as controls was 41.7 per cent.

If clinical data confirm the experimental evidence reported by Kay and Lockwood, a new and highly significant approach to the problem of appendiceal peritonitis will have been opened.

*Absorption from the Peritoneal Cavity.* The considerable experimentation directed toward determining how absorption from the peritoneal cavity occurs is unfortunately invalidated as Harvey and Meleney pointed out in a comprehensive review of the subject by two highly questionable premises: (1) that substances injected into the peritoneal cavity behave in a manner similar to fluids which are found in the cavity, and (2) that the method of absorption is the same in man and in animals. For these reasons an extended discussion of the subject seems unjustified in a purely clinical text and the reader is referred to the review mentioned for an excellent summary.

A continuation of these investigations (Kay, John H. Personal communication) indicates that two enzymes possess fibrinolytic activity rather than the single enzyme originally implicated. Their relative importance in appendiceal peritonitis remains to be clarified.



*Systemic and Visceral Changes* When the pathologic process in the appendix has advanced beyond the appendix and spreading peritonitis is established disturbances of fluid and chemical balance become an important part of the picture, their degree generally speaking being related to the stage of the pathologic process and the duration of illness. As a matter of convenience and to avoid repetition they are discussed in the sections dealing with the preoperative and postoperative care of complicated acute appendicitis (p. 309).

Advanced peritonitis if the patient lives long enough may affect all the viscera of the body and may give rise to irreversible changes particularly in such organs as the heart, liver and kidneys. Myocardial insufficiency is brought about by the action of bacterial toxins as well as by anemia and anoxia resulting from circulatory stasis. Wright and his associates who carried out special postmortem studies reported finding great amounts of blood stagnated in the splanchnic and mesenteric vessels and not available for the peripheral circulation. This observation explains the clinical manifestations of cyanosis, falling blood pressure and increases in the respiratory and pulse rate though the rising pulse rate is also a compensatory effort by the heart to carry to the tissues the greatly diminished amounts of blood it receives. The myocardial changes *per se*, are not the cause of the circulatory collapse. More speculative causes of the collapse include adrenal exhaustion and the depressant action on the medullary centers of toxins reaching them by nerve and other paths.

Changes in the pulmonary system according to Wright and his associates include edema, circulatory stasis, bronchitis, atelectasis, interstitial pneumonitis and pneumonia partly as the result of toxemia partly as the result of cardiac incompetence. High elevation of the diaphragm is a common result of the distention of the gastrointestinal tract which is usually present. Cerebral changes include edema and anemia of the brain as the result of toxemia, circulatory stasis and cardiac insufficiency. The liver and kidneys show the changes commonly associated with infection and cardiovascular insufficiency.

The causes of death in peritonitis are usually stated to be shock, intestinal obstruction and ileus, toxemia and cardiovascular collapse with terminal pneumonia playing some role. The statement of course is correct but in the endeavor to be accurate one should not lose sight of the fact that the ultimate cause of the fatality is acute appendicitis to which all other pathologic changes are secondary.

Kay and Lockwood's studies on induced appendiceal peritonitis in

## APPENDICEAL (PERIAPPENDICEAL) ABSCESS

*Pathologic Process* An appendiceal abscess has a more or less well developed wall the constituents of which as well as the location of the lesion depend upon the original position of the appendix. The most frequent site is between the cecum and the anterior abdominal wall though numerous other locations are possible (fig 40) and any structure other than the cecum such as other parts of the intestine the liver the omentum the mesentery the bladder or the pelvic organs may take part in its formation.

When an appendiceal abscess is adjacent to the cecum the purulent exudate is confined to the right flank right groin or retrocecal area. Ileocecal abscesses which because of their protected location are likely to be quite large before they are discovered are particularly dangerous for a number of reasons as Hicken and Carlquist have pointed out (1) Kinks adhesions and inflammatory edema of the ileum and cecum may precipitate an acute intestinal obstruction (2) If the suppurative process is in the neighborhood of the mesenteric vessels thrombophlebitis may follow with resulting segmental gangrene of the ileum or ascending pyelphlebitis (3) Deep seated abscesses may rupture spontaneously (as for that matter may an abscess in any other location) causing fulminating peritonitis (4) Operation is technically difficult because it is necessary to traverse a clean peritoneal space in order to evacuate the purulent exudate.

Hicken and Carlquist have placed pelvic appendiceal abscesses in four developmental categories (1) The diseased appendix hangs over the pelvic brim and its contents are emptied directly into the cul de sac (2) Pericecal infection soils the cul de sac before localization has taken place and two distinct abscesses finally form one about the cecum and the other in the pelvic cavity (3) A fistulous communication exists between the pericecal abscess and the abscess in the cul de sac (4) The collection of pus within the pelvis is part of a generalized peritonitis. Many observers of whom I am one would be inclined to consider the latter type a residual rather than a primary appendiceal abscess. For anatomic reasons the inflammation of a pelvic appendix extends first to the left. Then as the pelvis fills with purulent exudate the pus takes the path of least resistance which is alongside the sigmoid colon.

If the appendix is unusually long a left sided abscess may develop. If the appendix is so located that perforation occurs into the loose

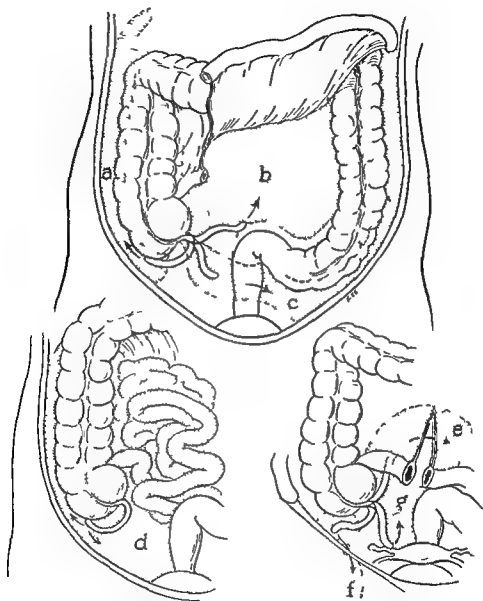


Fig 40 Possible locations of primary and residual appendiceal abscesses a extending upward toward subphrenic space b between medial aspect of sigmoid and mesentery of small intestine c on left side d extensive right sided abscess e mesenteric abscess f abscess pointing in thigh g ascending pelvic abscess

even in the absence of such drainage resolution may occasionally occur just as an inflammatory process can resolve anywhere else in the body. There is however no certain way of predicting in which cases this will happen and in which it will not.

Residual changes of one kind or another are likely to be present in any appendix that has been the site of a previous acute attack. Ulceration may so damage the mucosa and other parts of the wall that perforation is more likely to occur in a succeeding attack. Repeated attacks of bacterial appendicitis may cause permanent thickening of the wall. Healing by fibrosis may result in distortion of the lumen or division of the lumen by septa. Hyperplasia of the mucosa may occur. If the attack has involved the peritoneal coat of the appendix adhesions and bands may cause later attacks of obstruction and stricture formation within the lumen may have the same results. Occasionally the appendix may have been amputated by natural means and will be found free in the cavity while the perforation is sealed over. Such changes support Royster's concept that an acute attack of appendicitis particularly an attack in which gangrene and perforation occur within a few hours of the onset of symptoms represents an accumulation of pathologic changes of longer or shorter duration a sort of hoarded pathologic alteration rather than virgin pathology.

Three particular end results of recurrent attacks of acute appendicitis terminated by resolution deserve special comment namely obliteration mucocoele and diverticulum.

**Obliteration.** Total or subtotal obliteration of the appendix occasionally occurs as the result of recurrent acute attacks of appendicitis which terminate by resolution. Collins in a study of 1354 specimens (300 removed at operation 1054 collected from consecutive unselected autopsies) found that it was present in 29 per cent more often in the surgically removed organs than in the postmortem specimens obtained from comparable age groups.

Although obliteration was not observed until the seventh decade in more than half of the appendices studied more than a third were still patent in the ninth decade and the age range in totally obliterated cases from 12 to 96 years seems to dispose of the theory that obliteration is an involutionary process. It also makes it more reasonable to assume that after an attack of acute or subacute appendicitis which subsides by resolution the mucosa is partially or completely destroyed because the crypts of Lieberkuhn which are the source of mucosal regeneration cease to function. The inflamed portions of the lumen

areolar tissue behind the posterior cecal wall the abscess may extend upward to the subphrenic space or downward to the iliac crest, the tuberosity of the ischium or even the inguinal canal and the scrotum.

Appendiceal abscesses may be unilocular or multilocular the latter variety sometimes being formed by the fusion of several isolated pockets. In this connection it might be mentioned that supposed secondary or residual abscesses may be merely isolated primary foci overlooked during the initial exploration.

Following abscess formation the appendix or any segment of the appendix may project free into the abscess cavity. Sometimes the organ is so imbedded in inflammatory adhesions that it is difficult to determine just where it is located. Sometimes it is completely detached and floats free in the purulent exudate contained in the cavity, though more often it is still attached to its meso.

The larger the abscess the more omentum and intestine are involved in the process of localization and the greater is the risk that angulations and kinks will occur in the intestine with subsequent intestinal obstruction. On the other hand if abscess formation is occurring and if in the course of conservative therapy intestinal distention is not properly controlled the intestinal loops may become separated from the abscess and extension of the infection may thus occur.

Appendiceal abscesses sometimes rupture spontaneously into the bladder and other abdominal viscera (p. 236). They may enlarge upward and travel along the paracolic gutter to reach the subdiaphragmatic space (p. 213). They may extend posteriorly into the retroperitoneal tissues. They may travel downward beneath Poupert's ligament and point in the groin or on the surface of the thigh. They may point toward the umbilicus. They may communicate with sinuses on the abdominal wall. Finally and probably most important any appendiceal abscess may point toward and rupture into the peritoneal cavity. This accident can occur while the patient is under careful observation in the hospital. It is known to have happened in at least 4 cases in the Charity Hospital series with fatal consequences in all. The intra-peritoneal rupture of an abscess obviously has much the same effects as rupture of the diseased appendix into an unprepared peritoneal cavity.

### RESOLUTION

Naturally even if operation is not performed not every acute attack of appendicitis goes on to gangrene and perforation. A suppurative appendix with its lumen full of pus may drain into the cecum and



Fig. 42 Mucocoele of appendix visualized by barium enema (from Touro Infirmary New Orleans)

which are no longer protected by a mucosal lining then adhere to each other and later may become solidly fused.

The obliterated appendix is typically firm, stiff and noncollapsible. In spite of the thickening of the wall the organ is sometimes little more than a fibrous cord without a canal, though the process is not usually so extreme. In most obliterated appendices there is at the base, where the blood supply is more abundant than elsewhere, a small area of unaffected mucosa. The obliterative process usually begins at the tip and progresses toward the base, as would be expected because of the terminal character of the appendiceal blood supply.

**Mucocoele.** Mucocoele or mucoid disease (figs. 41A, B, 42) is a possible end result of the complete obstruction of an empty appendix. It may also occur when localized stenosis is of sufficient degree to seal off some part of the organ, or when mucus or pus is sealed into a portion of an appendix undergoing an obliterative process, particularly a process which originates at the base. The mechanism is simple. A closed cavity develops and mucus accumulates in it because secretion into the lumen exceeds excretion from it by drainage and absorption.

As a rule organisms gradually die out in the cavity and the contents are sterile; inflammatory changes are therefore not usual. Acute inflammation is, however, occasionally superimposed, as in the instance of the gangrenous appendix reported by Latimer in a 60-year-old man. Other changes are also possible. Thus Heatley reported a gangrenous hemorrhagic mucocoele with twisted pedicle in a 16-year-old girl, and

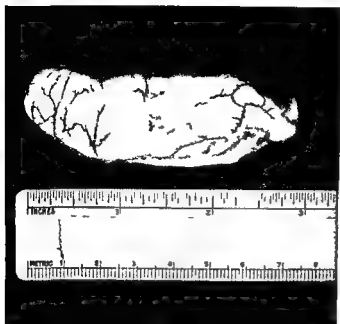


Fig 41A Mucocoele of appendix discovered in course of gynecologic surgery for another condition. The patient had never had symptoms referable to the appendix (courtesy of the late Dr Hilliard E. Miller)



Fig 41B Longitudinal section of appendix shown in fig 41A



Fig. 42 Mucocoele of appendix visualized by barium enema (from Touro Infirmary New Orleans)

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Woodruff and McDonald reported an instance of superimposed malignancy Bunch has called attention to the possibility of the deposit of mineral salts in the mucocoele and cited the case reported by Ogilvie of the railroad brakeman who complained of something solid tapping him inside whenever he was active and who was found to have a cyst of the appendix 2.5 by 1.5 inches with calcification of the walls\* The largest mucocoele on record seems to be the one reported by Sanders and Hundling in 1925 in a patient who presented a large tumor of the right lower quadrant the mucocoele measured 37 cm in its greatest circumference and weighed 450 gm He was perfectly well 18 years after its removal

To date according to Probstern and Lassar there have been reported in the literature 39 instances including their own case of mucocoele with myxoglobulosis In this type of mucocoele globoid bodies 2 to 3 mm in diameter and composed of mucoid material form clusters resembling frog eggs The condition is uncommon in clinical practice but is readily produced experimentally by ligation of the proximal end of the appendix The process apparently arises from a glandular injury but the pathogenesis remains to be fully clarified Probstern and Lassar point out that only 6 of the 38 cases reported in the literature were associated with symptoms referable to the appendix In their own case the mucocoele was responsible for cecal intussusception which required operation In the remaining cases the process was discovered at operation for other conditions or at postmortem

The greatest danger of mucocoele is rupture which is most likely to occur if growth is rapid and increasing intraluminal pressure causes thinning of the wall Since the viscid contents are practically sterile septic peritonitis is not the usual result of rupture though pseudo myxoma peritonei is a possibility Bunch reported 2 such cases both found accidentally at operation as well as 2 other cases in which pseudomyxoma peritonei of ovarian origin was associated with small perforations of appendiceal mucocoeles

Mucocoeles are not frequent Only 136 were seen at the Mayo Clinic in 43 000 cases of appendicitis studied by Woodruff and McDonald over a 24-year period and Jirka and Scudern who found only 4 cases

\* Another large calcified mucocoele of the appendix has been reported by Barnes [Barnes] Leyton Calcified mucocoele of the appendix *Am J Surg* 76 323 7 (Sept) 1948] It was detected by X-ray in a 79 year old woman but its true character was not determined until operation This patient also had cholelithiasis a diaphragmatic hernia a diverticulum of the ileum and multiple diverticuli of the sigmoid

in 9150 examinations at Cook County Hospital over an 8½ year period estimated the reported incidence as from 0.2 to 0.68 per cent.

*Diverticula.* Acquired diverticula of the appendix which are a possible result of previous acute inflammation of the appendix terminated by resolution must be distinguished from congenital diverticula which are exceedingly rare. The acquired type is the result of the development of stenosing scars and varying degrees of occlusion of the lumen as part of the process of resolution. Efforts of the muscularis to empty the occluded lumen usually lead to a marked increase in intraluminal pressure with occasional herniation of mucosa through areas of lessened resistance (muscular hirtuscs) in the muscularis. Bowers reported 2 cases in which diverticula were evidently the result of attempts by the appendix to expel fecoliths. Acquired diverticula are frequently multiple. The walls are composed of mucosa, loose areolar tissue and serosa in contrast to the walls of congenital diverticula which contain all the normal histologic components of the appendiceal walls. The absence of the muscular components provides a ready background for sudden perforation.

Collins, who reviewed the literature and reported 30 personally observed instances of diverticula of the appendix, found only 67 reported cases in 16,044 appendectomies. The largest proportion, 53 per cent, were located in the mid portion of the appendix and the next largest proportion, 28.3 per cent, in the tip. Approximately 40 per cent were multiple. Approximately 60 per cent were associated with acute appendicitis and approximately 28 per cent with perforation.

Collins' warning is entirely correct that if diverticula of the appendix are discovered in the course of surgery for other conditions, appendectomy should never be omitted, since subsequent acute inflammation is a serious possibility. The operation is the more justified because the signs and symptoms are likely to be atypical and early perforation is therefore frequent. Mucocèles are often associated with diverticula and their rupture into the peritoneal cavity may cause pseudomyxoma peritonei (p. 106).

### THE PROGRESSIVE PATHOLOGY OF ACUTE APPENDICITIS

Fitz's original description of the progressive pathologic process of acute appendicitis could scarcely be improved upon.

The inflammatory process once excited, its course and results show extreme variations. A simple catarrhal appendicitis is to be recognized anatomically but it is doubtful whether its clinical appreciation is possible. This appendicitis

in the absence of a concretion or foreign body may progress toward ulceration even to a peritonitis which may terminate fatally. In the presence of a foreign body or concretion these events are of likely occurrence. On the one hand the inflammation may result in the more or less complete obliteration of the canal of the appendix with or without circumscribed dilatation. On the other the ulcerative process becomes associated with a necrosis of the wall a peritonitis usually circumscribed at the onset and perforation. In those cases where the appendicular peritonitis represents the extension of an inflammation through the wall of the appendix without perforation permanent adhesions of the appendix to neighboring parts remain as evidence of the process. When it is associated with necrosis of the wall the inflammation of the peritoneal coat tends to become diffused and productive of serous and cellular exudations. The adherence of coils of intestine to each other and to the abdominal wall favors the accumulation of the exudation in a limited space and thus the formation of the tumor. At this stage the anatomical condition is a circumscribed peritonitis the appendicular peritonitis of With. This peritoneal abscess may then become absorbed or its contents may escape into the general peritoneal cavity through ruptured or softened adhesions. In the latter event is a rule death rapidly follows.

If the case does not terminate as thus stated the tumor may suddenly diminish in size with the discharge of pus from a hollow organ as the intestine bladder or vagina. The abdominal wall may become perforated and a sinus be established opening in the groin lumbar region or at the umbilicus. Such sinuses often remain open for a long time even many years.

The more protracted the course of the disease the greater is the probability of the destruction of the peritoneum forming the wall of the abscess. With the perforation of the parietal peritoneum may occur extensive necrosis purulent and fecal infiltration of the abdominal walls. Within three weeks the iliac muscle may be destroyed and the ilium be bared. The course of the psoas and iliacus may be followed into the thigh and extensive and deep seated destruction of tissue with fecal infiltration be present in this region. The pus may extend through the obturator foramen forming a deep seated abscess of the hip and thigh and may enter the hip joint.

The primary appendicular peritonitis may be continued into the tissues behind the caecum and thus a secondary pyatyphilitis or perityphlitic abscess be occasioned. So various are these possibilities that every case of so called perityphlitic abscess must be regarded as primarily one of a perforating appendicitis unless proven to the contrary.

With the frequent eventual destruction of the peritoneal wall of the abscess is the possibility of death from hemorrhage.

The occurrence of disease of remote parts may be alluded to as abscesses of the liver from pyelophlebitis or portal embolism in consequence of a mesenteric thrombophlebitis near the appendix. The affection of the liver and portal vein may be the result of a direct continuance of the phlebitis or may follow

putrid embolism from a thrombus in the immediate vicinity of the appendix. The extension of a secondary pyatyphilitis may cause perforation of the diaphragm with a consecutive pleurisy or pericarditis.

The following case history is an instructive example of the extensive and progressive pathologic process that can develop in a case of untreated acute appendicitis. The patient's history it should be noted pointed to a gastro intestinal disturbance.

**Case 4.** A white man 26 years of age 24 hours before hospitalization had pain about the umbilicus for about 30 minutes. Twelve hours later the pain reappeared in the same location and 7 hours later (19 hours after the onset) it became localized in the right lower quadrant. It was then accompanied by nausea and vomiting. The patient had had several previous similar attacks following dietary indiscretions and attributed the current illness to the same cause.

On his admission to the hospital the temperature was 98.6° F, the pulse rate 60 and the respiratory rate 20. Tenderness in the right lower quadrant was the only physical finding. The white blood cell count was 10,200 per cu mm, with 86 per cent polymorphonuclear leukocytes.

The initial diagnosis was gastro enteritis versus peptic ulcer. Treatment on a medical service consisted of intestinal decompression, infusions of glucose and physiologic salt solution and transfusion. Twenty four hours after admission the patient had a hard chill and 48 hours after admission the white blood cell count was 24,000 per cu mm, with 86 per cent polymorphonuclear leukocytes.

On the fourth day of hospitalization the patient had an acute attack of abdominal pain lasting about 4 hours. On the sixth day he had several milder similar attacks and a second chill. Rectal examination revealed generalized induration. The temperature at this time was 104° F, the patient was delirious and a uremic odor was noted. The nonprotein nitrogen of the blood was 42 mg per cent. The impression at this time was that a hollow viscus or possibly an abscess had ruptured into the peritoneal cavity and sulfanilamide therapy was instituted. On the eleventh day of hospitalization the patient was deeply jaundiced and extremely toxic. Several bedsores were present and spider angiomas were observed over the back. Death occurred 3 days later. The temperature had continued to range from 100 to 104.6° F.

Autopsy revealed a gangrenous ruptured appendix containing three fecaliths, generalized peritonitis, peri appendiceal abscess, multiple perforations of the large and small intestine, thrombosis of the branches of the superior mesenteric vein, pyelephlebitis, septic infarcts of the lungs and of the right lobe of the liver, pulmonary congestion and edema, bilateral broncho pneumonia, atelectasis of the right lower lung and bilateral pleural effusion, esophageal ulceration and septicemia.

## CLINICAL IMPLICATIONS

Acute appendicitis is evident from Fitz's description and from what has been said elsewhere as a potentially progressive disease and in many cases is a rapidly progressive disease. It should always be conceived of therefore from the standpoint of pathologic progression. It begins in all cases as a strictly localized intrinsic (intra-appendiceal) and therefore readily curable disease. It may terminate even if it advances beyond this stage by spontaneous resolution and retrogression. But if the disease does not thus terminate and if operation is not performed the pathologic process may continue until it involves directly or indirectly tissues or organs adjacent to and remote from the appendix. The disease may then be incurable by any means including surgery. The vestigial character of the appendix, its anatomic structure, its terminal blood supply, the possibility of obstruction and its potentially infected contents all make it clear why when once the disease process has been initiated the circumstances are all in favor of its assuming a progressively more serious character.

Two extremely important considerations arise from the acceptance of this point of view. (1) The time element in acute appendicitis is far more important than the precise pathologic stage at which the patient is seen, the virulence of the invading organism or anything else. (2) The wisest of clinicians and the most astute of pathologists cannot tell by any means whatsoever in any given case whether the process will subside spontaneously or will remain within the appendix or will go on to graver pathologic manifestations.

To these considerations should be added a corollary and a warning. The corollary, which might well be the slogan of every physician and every surgeon who treats acute appendicitis, is that if the worst has not already happened in a given case it may happen very soon. The warning is against attempts to estimate the pathologic stage likely to be present in any given case on the basis of the clinical picture or the time which has elapsed since the onset of symptoms. There is almost no disease in which pathologic process and clinical onset are precisely synchronous though it is generally true that the longer a disease has been evident clinically, and the more pronounced are the clinical manifestations, the more serious is the pathologic change likely to be. In acute appendicitis however the gravity of the conditions found at operation is often out of all proportion to the mildness of the symptomatology while the gravest pathologic complications may ensue within an un-

believably short time after the clinical onset of the disease, though they have not necessarily occurred within that period. The physician who bears these facts in mind is not likely to delay unduly in the diagnosis and treatment of suspected acute appendicitis.

The various stages of acute appendicitis are not sharply divided. They usually merge imperceptibly into each other pathologically as well as clinically. Theoretically each pathologic phase can be correlated with a corresponding clinical phase. Actually this correlation frequently does not occur. Terminal pathologic phenomena are sometimes present in association with clinical manifestations that suggest early phases of the disease. This anomalous state of affairs while it may be observed at any period of life is particularly frequent in patients advanced in years.

Opinions differ widely about the ability of clinicians to determine before operation the stage of the disease present in any given case of acute appendicitis. I feel very little confidence in my own personal ability in this respect. The Charity Hospital series of cases shows a high percentage of error in pre-operative attempts to determine the precise stage of pathologic change and many series in the literature show equally high proportions of error (p. 149). The part of wisdom therefore seems to be as already suggested to follow the plan advocated by numerous experienced surgeons and to work on the assumption that things are likely to be at least as bad as they seem if not worse. It is curious that writers who declare that they can tell what is happening within the abdomen in the majority of cases fail to emphasize the risk in the minority of cases in some reported series as high as 25 per cent in which they can make no predictions. By far the safer plan is to appreciate as Murphy did the degree of destruction that can occur in a very limited number of hours with virulent infection of the appendix.

Generally speaking gangrene is a more favorable development than perforation and localized peritonitis or appendiceal abscess is a more favorable development than spreading peritonitis. Yet none of these developments is really favorable and it speaks ill for our concept of acute appendicitis that we should so regard them. The whole matter is relative. Gangrene and rupture and peritonitis are properly regarded as complications of acute cholecystitis and other diseases and it is unfortunate that they are not similarly regarded in acute appendicitis. In this connection it might also be pointed out that if Royster's concept of acute appendicitis as a disease in which there have been pre-existing morbid changes is correct then the patient with an apparent first attack

is always a candidate for future attacks and prompt appendectomy would seem to be the procedure of wisdom

Finally this is the proper place to emphasize that the concept of the pathologic appendix should be based not only on what the pathologist finds in the specimen when he examines it in the laboratory but also on what the surgeon has to say about it at operation. Every surgeon of experience has observed case after case in which a perfectly typical history of acute appendicitis has made operation imperative but in which there are no gross changes in the specimen to account for the clinical findings. In other specimens reported negative in the laboratory it is perfectly possible to postulate and sometimes to demonstrate, an obstructive mechanism which given a little more time, would certainly have resulted in gangrene or rupture (p. 64). In this type of case there is really some justification for the surgeon who is given to remarking that if he had not operated when he did the appendix would probably have ruptured within the next few hours.

## ( VII )

### *The Clinical Picture and the Diagnosis of Acute Appendicitis*

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#### SYMPTOMS

Although Fitz's original description of the clinical picture of acute appendicitis must be read with certain reservations because it is derived from a series of cases in which perforation had occurred it is still essentially correct and it emphasizes as many modern descriptions do not the variable character and the absolute inconstancy of the syndrome. Sudden severe abdominal pain Fitz pointed out is the first and most constant symptom of the disease but thereafter nothing is constant. The pain may appear in the right iliac fossa hypogastrium umbilical region epigastrium hepatic region left iliac fossa or right hip and groin. It may extend upward to the liver or downward to the rectum testicle perineum or thigh. It may be preceded by diarrhea. It may be accompanied by nausea and vomiting or by fever which is seldom high or by chills or by disturbances of micturition.

#### PAIN

Pain as Fitz pointed out is the most constant and most important symptom and may be the only symptom of acute appendicitis though from that statement onward there can be no sweeping generalizations.

The pain usually occurs in the midst of perfect health. Often it wakes the patient from a sound sleep. Frequently it is of gradual onset and careful questioning often reveals that pain of even apparently sudden onset has been preceded for several hours or perhaps for several days by abdominal discomfort too vague to be described as pain and best expressed by the statement that a patient ordinarily unconscious of the functions of his abdominal viscera becomes aware of them. Graham's description of it as abdominal tiredness is another way of expressing it.

Very frequently the pain originates in the epigastrium or about the umbilicus but it may originate anywhere else in the abdomen. It may be pelvic right sided left sided bilateral or diffuse. It may extend to the rectum as Fitz pointed out or to the perineum or the thigh or



in males to the testicle usually the right testicle. It may also occur in the lumbar region or in the right shoulder. Some observers have stated that patients who have had one or more previous attacks are more likely to have the initial pain of subsequent attacks in the right lower abdomen but this is not a sufficiently general observation to warrant special emphasis.

At the onset the pain is usually colicky and intermittent but it may be continuous. In any event it is likely to be aggravated at intervals as the result of peristalsis. In the obstructive type of disease colicky pain is characteristic and may be very severe though the pain of acute appendicitis is practically never as severe as the pain of biliary or renal colic for instance or of mesenteric vascular occlusion. It is likely to be increased by changes of position sudden jars and pressure. Morley however observed that patients whose first pain is about the umbilicus sometimes find temporary relief by mild pressure and Finney Sr reported the case of a young boy with acute appendicitis in whom the diagnosis was greatly confused by his insistence upon constant massage of the right abdomen.

In the typical case of acute appendicitis at an interval varying from an hour or two to as long as 48 hours or more the pain regardless of its point of origin becomes localized in the right lower quadrant of the abdomen and is particularly severe over McBurney's point. Often however even if localization in the right side does occur it is not definite and sometimes particularly at the extremes of life it does not occur at all. When pain has once localized in the right lower quadrant subsequent extension to other parts of the abdomen should always be interpreted as indicating the development of some complication most often peritonitis.

There is no absolute correlation between the intensity of pain and the pathologic process and attempts to gauge the latter by the former are likely to end in disaster. The temperament of the patient and his personal threshold of pain chiefly determine the degree of his complaint. Negroes have a high threshold of pain which is one reason why such serious pathologic changes are often found in appendicitis in that race.

The cessation of pain if it occurs gradually may be an indication that appendiceal obstruction has been relieved or that spontaneous resolution is occurring though this outcome should never be deliberately waited for nor unless there is concurrent subsidence of other symptoms should it be regarded as indicative of recovery. It may also indicate the development of far more serious pathologic changes. If the cramping or spasmodic type of pain subsides abruptly gangrene or rupture may

have occurred the latter being a particularly likely possibility if the pain ends suddenly after a very severe bout

**Shoulder Pain** Mastin who first discussed right shoulder pain in acute appendicitis observed 2 cases in 11 years and collected 9 others 6 by a review of the American and foreign literature from 1920 to 1935 and 3 from replies to 351 questionnaires There was only 1 instance of shoulder pain in the 6441 cases of acute appendicitis studied at the New Orleans Charity Hospital over an almost 16-year period but this patient significantly lost his life undoubtedly because the unusual symptom confused the diagnosis

Shoulder pain does not develop until some hours after the abdominal pain From the diagnostic standpoint the important consideration is that its presence though distinctly unusual does not exclude the diagnosis of acute appendicitis Cope who observed 2 cases considered its possible occurrence a matter of sufficient importance to warrant routine inquiries concerning it the examiners hand being placed on the acromioclavicular region as the question is asked

**Origin of Pain** It is impossible as well as unnecessary in a book of this sort to enter into a full discussion of the origin of pain in acute appendicitis The immediate cause is twofold (1) stretching of the appendiceal wall which is composed chiefly of unstriated muscle fibers and (2) exaggerated peristalsis which is excited by obstruction due to various causes Upper abdominal pain is sometimes due to reflex pyloro-spasm but is more readily explained by the fact that the upper abdomen is the zone of distribution of the nerves that furnish the main nerve supply of the appendix There is not complete agreement with regard to sensory involvement in acute appendicitis the ninth tenth and twelfth dorsal nerves and the first and second lumbar nerves have all been implicated

Cope explained the testicular pain which sometimes occurs as due to irritation of the sympathetic nerve filaments accompanying the spermatic artery It seems more reasonable to regard it as referred pain since the testicle and the appendix are both supplied from the tenth spinal cord segment The testicular retraction occasionally observed may be accounted for by direct stimulation of the genitocrural nerve by inflammatory exudate

The shoulder pain of acute appendicitis is referred in Mastin's opinion by way of the phrenic nerve This is the only nerve with afferent fibers reaching the abdomen that also is connected with the cervical segment whose nerves (the third fourth and fifth) supply the area of pain The infrequency of pain in this location suggests that

the phrenic nerve in such cases has an anomalous distribution. Other possible explanations are that the cecum and appendix are abnormally high or that the appendix is in the ascending position and abnormally long. The explanation that the pain is due to an extension of the infection to the diaphragm obviously applies only to the cases in which such an extension has occurred.

#### GASTRO INTESTINAL SYMPTOMS

**Anorexia** Anorexia really the first degree of the symptom complex which includes nausea and vomiting is an important gastro intestinal manifestation which is usually overlooked. It was mentioned only 18 times always as a first symptom in the Charity Hospital series of cases which probably means that it was not usually inquired into. Incidentally, there were 3 deaths among these 18 patients. Cope suggested that a child suspected of having appendicitis be asked to name the last meal which he really enjoyed and the first for which he was not hungry and the same question might profitably be put to adults. Another profitable question to a person smitten in the midst of sound health is whether or not his acute illness has been preceded by a period of vague digestive discomfort which is more awareness of digestive processes by a person previously completely unaware of them than it is actual indigestion.

**Nausea and Vomiting** Nausea and vomiting are next in frequency to pain in acute appendicitis. Nausea is more frequent than vomiting but neither occurs constantly. In the Charity Hospital series of 6441 cases 54 patients did not complain of nausea 329 did not complain of vomiting and 329 (differently distributed) did not complain of either which means that approximately 11 per cent of all patients did not present the second most frequent of the so called classical triad of symptoms.

Vomiting is more likely to occur if the onset of illness is closely related to the taking of fluid or food or if fluid food or a purgative all of which excite gastro intestinal activity are taken after the onset of the pain. It usually occurs 3 or 4 hours after the initial pain but it may not occur for 12 to 24 hours afterward or even later. The vomitus at first consists of stomach contents later of bile stained fluid.

Vomiting in acute appendicitis is due to several possible causes. If it occurs early it is usually of reflex origin due to irritation of nerves of the peritoneum mesentery or stomach though it may also be due to obstruction of the appendix which is a tube composed of involuntary

muscle. In Cope's opinion vomiting at the onset of an attack of acute appendicitis means that the appendix is distended on the distal side of a stricture or other obstruction and that there is immediate danger of perforation. Vomiting late in the illness usually means an extension of the process beyond the appendix and in advanced cases it is probably due, at least in part to the action of absorbed toxins upon the medullary center. Constant vomiting in the opinion of some observers suggests that gangrenous changes have occurred.

It should be remembered in evaluating vomiting as a symptom of acute appendicitis that some persons vomit more readily than others particularly those prone to previous digestive upsets and those of marked reflex nervous susceptibility. Children are more likely to vomit in any illness than are adults.

*Constipation and Diarrhea* For a long time it was incorrectly stated that constipation was an almost constant symptom of acute appendicitis and the statement is still occasionally made. It is not true. Either constipation or diarrhea may be present or the bowel function may be perfectly regular and the physician who attempts to make a diagnosis on the presence or absence of any of these conditions is likely to be entirely wrong. Constipation however is significant if it occurs in a person accustomed to regular bowel movements if it has lasted for several days or if it is accompanied by flatulence or abdominal discomfort. The comment is fair that acute appendicitis should be diagnosed long before the patient not habitually constipated has had time to determine whether or not he has become so. Diarrhea due to purgation must be distinguished from diarrhea that is an integral part of the appendiceal syndrome. Early in the disease it may be caused by the pressure of an inflamed pelvic appendix on the rectum. Later it may be due to irritation of the rectum by a pelvic peritonitis or a cul de sac abscess.

The most important consideration about diarrhea is the diagnostic confusion to which it may give rise chiefly because it is not usually conceived of as part of the symptom complex of acute appendicitis. In the Charity Hospital series it was present at some stage of the illness in 266 cases 16 of which ended fatally. In 87 cases it was the first symptom. Diarrhea was also a first symptom in 3 of the 158 fatal nonsurgical cases studied at Charity Hospital and was present in the course of the illness in 15 other cases in this group. McClure and Altemeier stated that when it was present in their series of 252 perforated cases it delayed diagnosis from 1 to 7 hours.

Keyes has expressed the opinion that what he called "bowel con-

sciousness is always the initial discomfort in acute appendicitis. It is due to gas stoppage; it occurs only at the onset and in his experience a convincing story can always be secured spontaneously or in response to direct questioning except from children patients with a low intelligence quotient and those whose attack of illness begins with localization of pain. There is little or no relief from the passage of gas per rectum or from a bowel movement which is one of the reasons the patient is inclined to take a lavative.

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#### URINARY TRACT SYMPTOMS

Urinary symptoms and signs chiefly dysuria, frequency, pain and burning and gross and microscopic hematuria occur in a small proportion of cases of acute appendicitis and are not always easy to explain. If the appendix impinges on the ureter or the bladder the explanation is obvious but this does not always occur and the situation is further confused by the fact that in some instances in which urinary symptoms have not been noted autopsy shows the bladder to be involved in the inflammatory process. In the occasional case the appendix is adherent to the kidney. If the explanation were valid that the bladder during contraction pulls on the inflamed peritoneum it would seem that painful voiding should be more frequent than it is.

Collins found hematuria present in 124 of 1402 consecutive cases of acute appendicitis and in 104 of 751 cases of acute immobile retrocecal appendicitis. In 71 per cent of these cases the presence of hematuria was detected only microscopically. I am in complete accord with his interpretation of the importance of hematuria in acute appendicitis and would extend it to all urinary symptoms. The diagnostic confusion they introduce is what matters. In Collins' series operation was delayed on an average of 4 hours when hematuria was present and the case fatality rate was 62 per cent in that group of cases as compared with 47 per cent for the whole series. In the Charity Hospital series of 6441 cases urinary symptoms were present in 227 patients, 21 of whom (9.2 per cent) died as compared with the general mortality of 4.97 per cent for the whole series. Urinary symptoms were also present in 8 of the 158 fatal nonsurgical cases.

The following case is an illustration of acute appendicitis in which no suspicion of the primary disease was entertained because all the attention was centered on the urinary symptoms which were clearly accounted for by the position of the appendix.

*Case 5* A white boy 15 years of age suddenly began to vomit 72 hours before admission to the hospital. Twenty four hours later he had several hard chills and high fever. He complained of substernal pain worse on deep inspiration and of severe dysuria for 36 hours before admission to the hospital he had been able to pass only a few drops of urine. At the first examination the temperature was 103.4 F, the pulse rate 94 and the respiratory rate 22. Breathing was shallow. The abdomen was distended and there was slight tenderness just above the pubes and in the right loin. Five hundred cubic centimeters of urine were removed in the admitting room. Urinalysis showed microscopic hematuria but no other abnormal findings. The white blood cell count was 10,250 per cu mm with 92 per cent polymorphonuclear leukocytes. There was a marked shift to the left.

The admission diagnosis was pyelocystitis. Dysuria did not respond to routine measures and oliguria developed. The boy vomited almost continuously. On the second day of hospitalization he suddenly went into a state of shock. Examination at this time showed a friction rub on the right side and abdominal distention and rigidity. A diagnosis of ileus was made and routine measures for peritonitis including transfusion were instituted. On the following day the boy had a second shock like episode. At this time he complained of pain in the right lower quadrant of the abdomen where deep tenderness was now elicited. Following an infusion of 20 per cent glucose solution he passed 240 cc of urine examination of which revealed nothing to explain his condition. Death occurred shortly afterward.

Postmortem examination disclosed a gangrenous hemorrhagic appendix perforated in the distal third and generalized fibrinopurulent peritonitis. The appendix lay in the midline directly over the bladder.

### CHILLS

Chills which occur in a small percentage of cases of acute appendicitis are variously interpreted. Deaver stated that if they occur at the outset of the disease they are indicative of rapidly developing gangrene while if they occur on the second or third day and are accompanied by high fever and sweating they are indicative of metastatic or embolic abscesses. Several other observers share this view. Colp after an extensive study concluded that their presence is related to venous thrombosis and that there is a decided chance of the development of pyelophlebitis in such cases (p. 225). Other observers also share this view. In Colp's series of 2941 cases of acute appendicitis the incidence of chills was almost equally distributed among all types of pathologic change. My own opinion is that the chief importance of this symptom is like that of urinary disturbances the diagnostic difficulty it introduces. The Charity Hospital figures support this idea. Chills were present in

TABLE I  
ATYPICAL FIRST SYMPTOMS IN 6411 SURGICAL CASES OF ACUTE APPENDICITIS

	To 12 yrs Cases	To 12 yrs Deaths	13-39 yrs Cases	13-39 yrs Deaths	Over 39 yrs Cases	Over 39 yrs Deaths	Total Cases	Total Deaths
Pain	312	3	612	20	188	10	1112	33
Right lower quadrant	7		26	1	4		37	1
Left lower quadrant	1		13		1		15	
Right groin	24		40		10	9	74	9
Right leg	2		41	1	8		51	1
Right costal margin	18	10	66	9	22	17	106	36
Nausea or vomiting or both	29	1	33		5	1	67	2
Headache	100	1	87	1	17	2	204	1
Fever	17	2	22	1	6	1	45	1
Malaise	5		23				28	
Backache			9		1	1	10	1
Flatulence	26		40	1	21	2	87	3
Diarrhea	2	2	11	1	5		18	
Anorexia	9		22		13		49	
Chills					2		2	
Hematemesis								

TABLE 2  
 ATYPICAL SYMPTOMS AND SIGNS (NOT INITIAL) IN 641 SURGICAL CASES OF ACUTE APPENDICITIS\*

	T, 12 yrs Cases Deaths	13-19 yrs Cases Deaths	Over 19 yrs Cases Deaths	Total Cases Deaths
Pain only symptom	25	11	20	128
Lumbar pain	2	1	2	26
Pain right leg	78	6	16	30
Left-sided pain	11	3	11	4
Right shoulder pain	3	1	1	4
Scrotal pain	1	1	1	6
Urinary symptoms	61	7	32	21
Diarrhea	28	6	31	179
Chills	28	4	32	210
No elited	1	1	1	3
Melenia	4	1	2	6
Blackcho	3	23	1	28
Syncope		13	1	14
Hiccoughs		2	2	4
Cervical tenderness		20	1	21
Vestigo		9	1	10
Projectile vomiting		2		2
No nausea and/ r vomiting	101	6	101	409
No localization	24	7	41	89
Left localization			1	1

\* The list is not complete



259 of the 6 441 cases studied and the mortality in this special group was 85 per cent (22 cases) as compared with a mortality of 4.97 per cent for the whole series. Chills were also present in 18 of the 158 fatal nonsurgical cases which is further proof of this point of view.\*

#### OTHER SYMPTOMS

Pain, nausea and vomiting it is agreed are the most frequent symptoms of acute appendicitis and most frequently appear in the order listed. Intestinal disturbances, urinary symptoms and chills are observed in a small but quite definite proportion of cases. In addition a wide variety of other symptoms may be observed at the onset of the illness or during its course apparently as part of the appendiceal disease. Cases of acute appendicitis complicated by concurrent disease are not included in this observation. The statement has been challenged but seems justified by the facts that in addition to the more usual symptoms almost any other conceivable symptom may be present and that both usual and unusual symptoms may occur in any conceivable order. The surgical series of cases studied from the New Orleans Charity Hospital (Tables 1-2) proves that point and in the 156 nonsurgical cases analyzed from the same institution unusual symptoms and signs and bizarre combinations were even more frequent proportionately than in the surgical group and obviously had more generally disastrous consequences.

The diagnostic difficulties introduced by unusual clinical syndromes are evident in the mortality rates associated with them. If these atypical possibilities are not constantly borne in mind appendicitis may not be thought of as a diagnosis or diagnosis may be so long delayed that the patient's life is jeopardized by attempts to establish it.

The Charity Hospital observations are substantiated and supplemented by the observations of others. Fifty-nine per cent of the 252 patients with perforated appendices studied by McClure and Altemeier had atypical histories. In 62 of the 1 000 complicated cases studied by Totten pain was not the first symptom and the mortality in this group was 40.3 per cent. In 55 cases rigidity was not present and the mor-

\* In a recent histologic study of 100 consecutive acute appendices [Remington, John H. and McDonald, John R. Vascular thrombosis in acute appendicitis. *Surgery* 24: 787-92 (Nov.) 1948] Remington and McDonald found evidence of thrombosis in one or more vessels in 26 specimens. There were 15 instances of gangrene and 5 of rupture in this group and fecaliths were present in 13 specimens. It was the authors' opinion that thrombosis would probably be found in most gangrenous appendices if serial sections were examined and they note that phlephlebitis may have its origin in appendiceal thrombosis though there was no relation between the findings and the postoperative course in this series.

tality in this group was 29 per cent. Arnheim and Neuhof stated that in their experience patients with acute appendicitis only rarely presented atypical histories but the comment seems warranted that their experience is itself atypical.

The literature abounds with histories of atypical cases of which a few samples may be given.

Bryant in 1887 reported a case characterized by uncertainty of diagnosis and by the unusual measures [appendectomy and drainage] taken for relief. A 45 year old white man developed diarrhea for which he took a cathartic with some relief. Ten hours later he had mild epigastric pain which he attributed to the preceding intestinal disturbance but 5 hours later when it became worse he called his physician who prescribed another cathartic. The pain increased tympanitis developed and vomiting ensued. Fifty hours after the onset on the advice of three consultants exploration was undertaken it revealed a gangrenous appendix perforated in three areas and generalized peritonitis.

Stevenson reported the case of an obese 41 year old woman with a huge panniculus who had had attacks of lower lumbar brackache several times yearly for three years. Three days before she was seen in her current attack she had begun to have the same type of pain. Examination revealed deep left iliac tenderness. The temperature was 104° F. The white blood cell count was 40,200 per cu mm with 78 per cent polymorphonuclear leukocytes. 25 per cent of the cells were young. On the fourth day of illness the patient complained of pain in the right lumbar region about the kidney. There was no response to prontylin therapy and death occurred on the seventh day of illness. Autopsy revealed a gangrenous and ruptured appendix lying extraperitoneally and extending over the right iliac artery across the posterior aspect of the true pelvis into the sigmoid mesentery. The infection had spread into the aortic glands on both sides of the vertebral column through the lymphatics of the trunk and on the left side every gland was a pus sac. There were also multiple abscesses in the perirenal fat. The usual operation for appendicitis in the author's opinion would have transformed the retroperitoneal infection into a fatal generalized intraperitoneal infection.

Barber reported the case of an obese 46 year old white woman who developed symptoms of acute appendicitis which was treated by the Ochsner Sherren method. Twenty four hours later she developed violent mental symptoms tried to throw herself out of the window and required several persons to restrain her. Four days later she was certified and transferred to an institution for mental diseases completely disoriented and quite maniacal. Here the abdomen was opened and a



Fig 43 Well visualized appendix following air contrast enema (case 6)

gangrenous appendix removed a pelvic peritonitis was present and the cavity was drained. Surgical convalescence was satisfactory and the patient within 3 weeks of operation regained her normal mental state. She had no recollection of the period of acute mania.

A particularly unusual case is reported through the courtesy of M. E. DeBailey.

**Case II** A 40 year old white woman experienced vague generalized abdominal pain followed 48 hours later by melena apparently originating in the large bowel which lasted with increasing severity for 3 days during the same period the abdominal pain also increased in severity. Physical examination revealed only right sided tenderness more marked in the lower quadrant. The white blood cell count was 10 000 per cu mm with 80 per cent polymorphonuclear leukocytes. Other laboratory examinations were essentially negative. A barium enema on the sixth day of illness after rectal bleeding had become less showed a definite rather extensive filling defect in the cecal region and ascending colon just below the hepatic flexure whether it was of malignant or inflammatory origin could not be determined. For the next 2 days pain and tenderness were more definitely localized in the right lower quadrant where a mass seemed to be developing. The diagnosis of acute appendicitis was not entertained because of a history of appendectomy for acute disease and the presence of a McBurney scar.

On the eighth day of illness edema was observed in the right lower extremity and within 48 hours had involved the whole limb. On the tenth day purpuric areas of discoloration were observed over the toes and extensive edema appeared on the left side. Lumbar sympathetic blocks carried out on

the tenth and eleventh days of illness were apparently not effective and the areas of discoloration became larger and deeper coalesced and eventually presented the typical picture of early gangrene. Gangrenous areas appeared on the left side on the fourteenth day of illness. The appearance of both limbs was somewhat suggestive of phlegmasia alba dolens. Therapy up to this time included only penicillin which had to be discontinued after 72 hours because of a skin rash and several transfusions of plasma and whole blood.

The assumption at this time was (1) that the abdominal lesion was inflammatory though malignancy could not positively be excluded in the absence of exploration, (2) that it had precipitated a severe thrombophlebitis probably of the iliofemoral region with later extension to the iliac vein on the right and still later involvement of the same regions on the left and (3) that a very severe arteriospasm had affected the lower leg and foot and that subsequent arterial thrombosis in the lower vascular channels accounted for the development of gangrene in these areas.

On the fifteenth day the patient experienced for the first time sharp pain in the right chest and roentgenographic examination showed increased density in the right lower base. Subsequent serial studies pointed more and more definitely to the diagnosis of pulmonary infarction.

The patient gradually improved under a regimen of penicillin transfusions and the usual supportive measures. Edema which had eventually extended to the level of the umbilicus gradually subsided and areas of gangrene began to demarcate. The temperature which had been about 100° F gradually fell to normal. Barium enema on the fifth day of illness showed the cecum contracted to a narrow tubular channel with the constricted segment sharply demarcated from the apparently normal bowel. A tentative diagnosis of ameboma was made and emetine was given for 10 days. At the end of this time another barium enema (fig. 43) showed the same findings and also showed a fairly well visualized appendix.

Exploration of the abdomen a day or two later revealed the cecum bound down by relatively recent adhesions in which the appendix was included and from which it was freed with some difficulty. It was retrocecal and perforated at the base. At the site of perforation was a small well walled off abscess. The appendix was removed and the defect in the cecum at the appendicocolic junction was repaired. Exploration of the pelvic area showed a normal arterial tree. The iliac veins however were completely thrombosed on both sides and were represented only by fibrous cords up to their junction with the vena cava and as far distally as palpation was possible. A specimen excised for examination from the vein on the right was found completely filled with a well organized thrombus.

The patient eventually made a complete recovery complicated by the development of a localized pericecal abscess which required drainage. After the gangrenous areas on the lower extremities had become fully demarcated the

large toe and the second toe on the left foot and all of the toes and part of the dorsum of the foot on the right were amputated

The vascular manifestations in this case almost completely overshadowed the comparatively mild symptoms referred to the abdomen. The picture was further confused by the presence of melena and by the misleading history of a previous appendectomy. The case furnishes one more proof of the importance (1) of informing a patient precisely what has been done at operation and (2) of prompt interval removal of the appendix after conservative treatment whether surgical or nonsurgical, of complicated acute appendicitis.

### VITAL SIGNS

*Temperature* Attention has already been called to the great disservice done by Murphy's insistence on a temperature elevation as a cardinal sign of acute appendicitis (p. 13) and might well be called to Fitz's wiser observation that fever may accompany the disease. If an attack of acute appendicitis is not interrupted by surgical interference or does not resolve spontaneously, fever would be expected sometime during its course just as it would be in any other inflammatory process in which bacterial invasion ultimately occurs, but it is almost never present early in the obstructive type and it is frequently absent early in the infectious type. In the occasional case (Table 1, p. 120) it may be the first sign of illness. Generally speaking in the usual case of acute appendicitis which has progressed for 10 hours or more there is likely to be a mild temperature elevation and again generally speaking the longer the disease lasts the more likely is fever to be present. The temperature may remain normal however until rupture has occurred.

Though the temperature is more likely to be low than high in acute appendicitis it may be of any level from extreme hyperpyrexia to subnormal. It is likely to be higher in young children than in adults and is usually much lower in aged subjects who react sluggishly to inflammatory processes. A sudden fall may indicate perforation or gangrene and the development of very high fever usually indicates impending or actual peritonitis. Royster stated that in his experience acute appendicitis ushered in with high fever has a grave prognosis. It is not possible however to correlate the temperature elevation with the pathologic stage. In the Charity Hospital series 31.5 per cent of all patients had normal or subnormal temperatures or elevations of less than 99° F when they were first seen.

*Pulse* Elevations of the pulse rate tend to occur with greater regularity and with more constancy than temperature elevations though the rate may be perfectly normal or even lower than normal (assuming 72 to be the normal rate) In the Charity Hospital series almost 45 per cent of all patients had subnormal or normal pulse rates or rates of less than 90 when they were first seen

When the fever is high and the disease is advanced the rate is likely to be correspondingly rapid though this is not always true It is quite as impossible in fact to make categorical statements about the pulse rate in acute appendicitis as it is about the temperature elevation Generally speaking young children exhibit faster rates than adults and older subjects because of the sluggishness of their reaction to disease exhibit the slowest rates Bradycardia has been observed in some cases and is usually believed to be associated with the gangrenous variety of appendicitis Bernstein and Goldsmith however who reviewed the literature in reporting a case emphasized that it may also occur in nongangrenous disease and pointed out that its real importance lies in the realization that it may be present a realization which they correctly state may be lifesaving

*Respiratory Rate* The respiration in early acute appendicitis is usually within normal limits or only slightly elevated though it may be altered if the abdomen is voluntarily splinted It is always faster and shallower when the appendix has ruptured and peritonitis has developed particularly if there is extension of infection to the diaphragm In children the respiration is normally much faster than in adults and in aged subjects it is frequently slower My impression which I cannot substantiate by statistical data is that the prognosis is poorer in patients whose respiratory rate is very rapid when they are first seen

*Blood Pressure* Alterations in the blood pressure are not present in either typical or atypical cases of acute appendicitis unless sudden rupture of the organ or of an intraperitoneal abscess occurs Then a fall in the pressure is likely to occur just as it would occur in any other instance of visceral rupture

## PHYSICAL FINDINGS

*Posture and General Observations* A patient with acute appendicitis if seen early in the attack may be ambulatory If the pain is severe he may walk with a limp and bend forward Sometimes he is literally doubled up A patient seen late in the attack may also be ambulatory Fitz observed a sailor who worked for 13 days after the onset of symp

toms and entered the hospital only 24 hours before he died of a gangrenous appendix with generalized peritonitis. He also mentioned the case reported by Buck of a sailor with acute appendicitis who continued to roll heavy barrels of flour until he entered the hospital with a prominent iliac tumor extending along the outer half of Poupart's ligament.

Such cases are not common. More often the patient will be found in bed even early in his illness. He may appear uncomfortable but he does not usually look very ill. If he lies supine he may prefer to flex one or both knees, sometimes almost on the abdomen. If he lies on the right side he is likely to assume the jack-knife position. He does not usually wish to lie on the left side for traction on the attachments of the appendix increases the pain, the organ being displaced as the cecum drops inward. He may lie quietly if the pain is constant but if it is colicky as it is in the obstructive type he may toss about as does any other patient with colic. He seldom occupies a position of rigid immobility as does the victim of a ruptured peptic ulcer or of acute pancreatitis.

Examination of the tongue—which is not the habit of many modern physicians—usually reveals it to be slightly coated or less often heavily coated. The breath may be foul.

Inspection of the abdomen does not usually reveal much early in the disease. The normal respiratory movements may be limited because the patient voluntarily splints his muscles. Auscultation also does not reveal very much unless a purgative has been taken and hyperperistalsis is present.

**Local Tenderness.** Local tenderness over the site of the appendix is the most constant physical finding in acute appendicitis though sometimes it is masked by generalized pain which has not yet localized. The tenderness appears to be located in the appendix itself for its site often seems to vary according to the position of the organ. On the other hand since viscera are themselves insensitive it is clear on reflection that the tenderness must be due either to cutaneous hyperalgesia (p. 131) or to local peritoneal irritation from stimulation of the subserous nerve plexus connected with the cerebrospinal system or to both causes. The nerve plexus is unduly sensitive because it is directly affected by extension to it of the appendiceal irritation.

Although localized tenderness is often associated with tenderness over the whole iliac fossa it is usually possible to find a single point of maximum tenderness which is most often the point described by McBurney (fig. 44). Since the description of this point is so universally

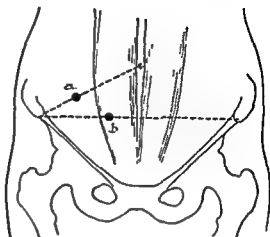


Fig 44 a McBurney's point b Lanz's point

misquoted it may be well to repeat it here. It is the seat of greatest pain *determined by the pressure of one finger* very exactly between an inch and a half and two inches from the right anterior spinous process of the ilium on a straight line drawn from that process to the umbilicus. The point remains fixed it is claimed no matter what the position of the appendix may be because it represents not the position of the organ but the point on the anterior abdominal wall at which the nerve endings of the eleventh and twelfth dorsal segments are reflexly irritated by appendiceal inflammation. It naturally will not be present if the appendix is retrocecal or pelvic or if it lies in a fossa on the posterior abdominal wall.

All observers do not agree that the point of maximum tenderness is the point described by McBurney. According to Cope it is usually a spot just below the middle of the line joining the anterior superior spine of the ilium and the umbilicus which corresponds roughly with the base of the appendix. According to Lanz (fig 44) it is located at the joining of the right and middle thirds of a line drawn between the anterior superior spines of the ilium. According to Clado it is at the intersection of the right and left anterior superior spinous processes and the lateral border of the right rectus muscle. According to Gray it is located about an inch and a half below and to the right of the umbilicus, the tender spot possibly corresponding to the point of emergence of the terminal branch of the eleventh dorsal nerve.

Attempts have also been made to identify the actual position of the appendix by various methods. Hertzler suggested that the base of the appendix corresponds exactly with the common apex of three equilateral triangles within a larger triangle formed by lines drawn first from the



umbilicus to the anterior superior spine of the ilium second from the umbilicus to the tubercle of the pubis and third by a line joining the spine and the tubercle. Munturborn contended that tenderness is more frequent over the lower end of the cecum because the mid inguinal position of the appendix is most frequent. With pain in the lower abdomen and tenderness just a little to the right of the median plane the appendix in his opinion is likely to be hanging over the pelvic brim while in the high retrocecal type of origin tenderness might be above the anterior superior iliac spine and in the prerenal variety it might be as high as half way to the costal margin. Manning stated that he had localized the acute retrocecal type of appendix in hundreds of cases by eliciting tenderness by deep pressure at a point just internal to and above the anterior superior iliac spine, as pointed out by some forgotten French surgeon. Such descriptions could be multiplied.

**Rigidity** Rigidity of the muscles overlying the appendiceal region is a frequent though neither a constant nor an essential sign of acute appendicitis. It is almost always found in young persons whose nervous systems react readily to stimuli of every kind but it becomes less constant with advancing age and it is frequently entirely absent in persons of advanced age even when extensive peritonitis is present. It is likely to be absent in persons of poor muscular development and in obese persons whose muscular fibers may be so intermingled with fat that rigid contraction cannot occur. It varies with the degree of sensitivity of the individual nervous system, the degree of parietal peritonitis, the localization of the infection, the associated congestion, the proximity of the appendix to the abdominal wall and the involvement of the nerve endings. Cope has pointed out that a muscle cannot maintain a constant contraction beyond a certain point at which the neuromuscular reflex becomes fatigued and that the absence of rigidity in any given case of acute appendicitis can sometimes be explained on this basis. The reflex may also be affected by absorption of bacterial poisons, a patient with peritonitis often being so intoxicated that tenderness and rigidity are both absent. Dvuzhlnaya explained muscular rigidity as a form of myositis caused by the entry of micro-organisms or toxins into the abdominal wall, a process facilitated by close contact between the primary focus of infection and the parietal peritoneum. The most usual explanation is that it is due to peritoneal irritation.

Work of the Mayo Clinic using comparative tensionometric readings carried out a study of abdominal rigidity which indicated that the degree is usually the same on both sides regardless of the cause of the intra abdominal pathologic process and in spite of clinical evidence to

the contrary. His explanation was that since the cæteron is embryologically a midline organ rigidity caused by visceromotor reflexes would be expected to be bilaterally symmetrical. Priestley, discussing this contribution remarked that the general belief in unilateral rigidity is an example of how a clinical impression can be handed on from generation to generation without question of its accuracy. He advised that both sides of the abdomen always be palpated simultaneously and that the defensive mechanism should be regarded as more significant when it involves the entire abdominal wall than when it is localized over the inflamed tissues. These observations are included at this point merely to complete the discussion so far as I know they have not been corroborated or even investigated by other workers.

**Hyperesthesia.** Local hyperesthesia is a state of increased sensibility to touch is not a constant finding in acute appendicitis but is a useful diagnostic aid when it is present. It was first observed in 1903 by Sherren who regarded it as dependent chiefly upon the degree of appendiceal distention which explains why it is most often observed in cases without perforation. It may however be present in any other variety and has also been observed in other intra-abdominal diseases. In patients who are not operated on and whose disease subsides by resolution cutaneous hyperalgesia disappears and it may be the last remaining finding in patients who are seen when resolution is almost complete.

Sherren defined the area of hyperalgesia in acute appendicitis as varying

from a complete band extending on the right side from the middle line below the umbilicus in front to the lumbar spines behind down to a small circular spot a little above the middle point between the umbilicus and the anterior superior spine. This band corresponds to the eleventh dorsal area of Head. The normal area is of a triangular shape. Its lower boundary reaches almost to Poupart's ligament its inner almost to the middle line and its apex is a little outside of the anterior superior spine sometimes extending to the mid axillary line.

Variations are possible. Occasionally the area is bilateral and in very occasional cases hyperalgesia appears on the left side. Levitas who observed mild hyperalgesia on the contralateral side in 95 per cent of his proved cases of acute appendicitis explained the contralateral reflex as due to apparently crossed conduction pathways of afferent components of the cerebrospinal nerves. It will be observed that the area affected lies in the area of distribution of the nerves from the tenth eleventh and twelfth dorsal spinal segments which means that hyperal

gesia remains substantially in the same location regardless of changes in the position of the appendix.

*Distention* Distention whether localized or generalized is an inconstant sign in early crises of acute appendicitis and is not a constant sign in advanced disease though it may be prominent in cases of ileus and of diffuse peritonitis. It is a fairly frequent accompaniment of acute appendicitis in patients over 40 years of age in whom it often leads to a diagnosis of intestinal obstruction. It is particularly likely to be present when the appendix is retrocecal and embedded in the cecal wall. It may be caused by the presence in the intestine of either gas or fluid in excessive quantities. Gradual distention of the cecum and terminal ileum is suggestive of a localizing process while the development of generalized distention indicates that the disease is spreading. Borborygmi, tympanites and sometimes painful peristaltic waves may accompany distention but distention may be present without other symptoms or findings.

#### LABORATORY DATA

*Blood Studies* The most useful and most generally practiced laboratory method in acute appendicitis is a complete blood count. A number of observers beginning with Sondern (cited by Royster) in 1905 have studied the leukocytosis and polymorphonuclear percentage and have drawn up diagnostic criteria on the basis of their observations. As generalizations the criteria are probably substantially correct. As guides for diagnosis in the individual case they are unreliable and misleading and there has been for many years a growing tendency to disregard the findings altogether. It is said that A. J. Ochsner taught his students to have blood counts made routinely but not to look at them until after the appendix had been removed.

It is frequently stated that the average leukocytosis in acute appendicitis is between 10,000 and 12,000 per cu. mm., but how misleading such generalizations can be is evident from the analysis of the Charity Hospital cases. Neither the white blood cell count nor the polymorphonuclear leukocytic percentage which is usually regarded as more reliable than the total count showed any consistency in respect to pathologic process or to the outcome of the disease. In the series of surgical cases more than 25 per cent of all patients had leukocyte counts lower than the level usually regarded as the average for the disease.

Other reports bear out these observations. Mason and his associates found that 25 per cent of the patients with unruptured appendices in

their series and more than 10 per cent of those with peritonitis or abscess had leukocyte counts under 10 000 per cu mm Johnson in a careful analysis of 221 cases in which the diagnosis of acute appendicitis was entertained concluded that when both the total and the differential white blood cell count are abnormal (over 10 000 per cu mm and over 70 per cent respectively) the chances are that the disease is acute appendicitis but that neither the one finding nor the other can safely be used to diagnose or exclude the disease In fact if these criteria had been used for the decision for and against operation in his personal series a number of patients who needed surgery would not have been operated on while a number of others with nothing at all the matter with them would have been submitted to surgery

The blood findings may be affected by circumstances unrelated to the appendiceal disease McKenna in 1919 called attention to the low leukocytic count as well as the unusually mild clinical symptoms in acute appendicitis after injection of triple typhoid vaccine Some 40 per cent of 200 subjects entirely normal or with only minor ailments had white blood cell counts of 7 000 or lower after such injections so that in them a count of 9 500 would represent a relative leukocytosis to a physician aware of the circumstances but might be dangerously misleading to one who was not Somewhat similar observations were made by medical officers in World War II and have also been made in measles and in certain virus diseases

*The Schilling Hemogram* Studies of the white blood cell count are generally though not universally believed to have become more useful since the introduction of the Schilling hemogram which divides neutrophilic leukocytes into four groups namely myelocytes juveniles stabs and segmented forms The index is a ratio of the total shift or immature cells (i.e. myelocytes plus juveniles plus stabs) to the segmented forms A shift to the left of the nonsegmented cells is regarded as indicative of acute disease

There are various modifications of the original Schilling hemogram There are also many reports of the possibilities of the method in relation to exactness of diagnosis In Warmock's series for instance the percentage of immature leukocytes was increased in proportion to the gravity of the pathologic changes Carlson and Wilder also found a general correspondence between the degree of shift from normal and the progressive degree of the appendiceal disease process There were however so many variations within each group that it was recommended that no attempt be made to differentiate degrees of pathologic change by this method but merely to differentiate inflammatory from

noninflammatory conditions. These same authors felt that a marked shift to the left indicates a bad though not necessarily a fatal prognosis. Some of their patients who survived showed greater shifts than those who died. Crocker and Valentine, who modified the Kohl modification of the original Schilling method, recognized eight degrees of pathologic change in the appendix by the use of the whole hemogram. Jennings and his associates did not find that hemography yielded sufficient information as compared with the usual differential count to warrant its use and characterized the method as time consuming and operation delaying. My own conclusions are to much the same effect. The Schilling hemogram may be useful in correlation with other observations but it should not be relied upon alone and it is not of sufficient value to justify any great delay in operation while it is being carried out.

**Blood Sedimentation Time.** A study of the blood sedimentation time has been suggested as a method of differentiating acute appendicitis from pelvic inflammatory disease, the rate being supposed to be much more rapid in the gynecologic conditions. Schockaert and his associates\* who found the method reliable in 95 per cent of their cases stated that if at the end of an hour the rate has attained or exceeded 20 mm. it may be assumed that the condition is pelvic inflammatory disease and operation may safely be withheld. Lintgen and Fry found diagnosis by this method accurate in 27 of 30 cases of pelvic inflammatory disease but incorrect in 48 of 100 cases of acute appendicitis. Collins found the test reliable from the gynecologic standpoint in 96.3 per cent of the cases in which it was used but incorrect in almost a quarter of a series of supposed cases of salpingitis, all of which were instances of perforative appendicitis with varying degrees of peritonitis. My own opinion is that the sedimentation rate is dangerously unreliable in the diagnosis of acute appendicitis and I see few indications for its use.

**Other Laboratory Studies.** Urinalysis should be performed routinely in the diagnosis of acute appendicitis to eliminate renal disease and diabetes. Blood chemical studies should be carried out if they are indicated but in the absence of diagnostic difficulties and special indications they do not form part of the diagnostic regimen of the disease. Roentgenologic examination of the abdomen by a flat film is also carried out if indications are present which they may be in advanced cases but in the early case the method is not of value except

\* *Diagnosis of salpingitis and appendicitis (Foreign Letters) J A M A 112*  
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to exclude biliary and renal (ureteral) calculi. Examination of the gastro intestinal tract with a barium meal or a barium enema is absolutely contraindicated in patients with suspected acute appendicitis. Roentgenologic examination of the chest is a valuable measure in cases in which the diagnosis lies between appendicitis and early central pneumonia.

### OBSTRUCTIVE ACUTE APPENDICITIS

Although the obstructive type of acute appendicitis had been mentioned by van Zwalenburg in 1905 and fully described by Wilkie in 1914 the latter author wrote in 1931 that patients with this highly fatal type of disease were not then being operated on any earlier than they had been 20 years before. Even today when Wangensteen and his associates have supplied full proof of both the frequency and the seriousness of obstructive acute appendicitis there is still no general comprehension that it even exists.

In this type of disease Wilkie pointed out the essential clinical feature is the sudden onset of intermittent colicky pain, sometimes associated with vomiting without an appreciable rise in temperature or pulse for a period of some hours. If the appendix is empty at the onset the underlying pathologic process may be relatively mild. If it contains a foreign body, such as a fecalith or even feces, rapid distention is likely to occur, tension gangrene of the wall follows, and within a short time, sometimes not more than 12 or 18 hours, perforation occurs into an unprepared peritoneal cavity. Often perforation is hastened by the administration of a purgative in the belief that the condition is intestinal colic. Obstructive appendicitis Wilkie concluded is a variety of disease in which the difference of a few hours in treatment may also make the difference between fairly certain recovery and the risk if not the certainty of death.

Wilkie's clinical observations have been clarified by the pathologic demonstrations of Wangensteen and his associates. The clinico-pathologic process according to these observers is substantially as follows. The appendiceal lumen, regardless of the method of occlusion, forms a closed loop. Peristalsis is stimulated as the appendix endeavors to overcome the obstruction and sudden severe cramping pains occur. Peristalsis and obstruction stimulate secretion from the mucosa, and the lumen gradually fills with fluid, the quantity eventually being increased by bacterial action. Until the bacterial factor is introduced, temperature, pulse rate, and leukocytic level remain actually or relatively unchanged.

As distention within the lumen of the appendix begins to cause pressure on the terminations of the sympathetic nerves the pain which up to this time has been colicky becomes more or less constant in character and is referred to the umbilical region. With increasing distention the capillaries and venules of the appendix become occluded and vascular congestion, edema and diapedesis of leukocytes are the next step because the blood continues to be pumped into the arterioles at systolic pressure. When distention reaches such proportions that reflex nausea and vomiting occur the right lower quadrant is clearly recognized as the origin of the pain which continues until distention and inflammatory reaction so increase that the terminations of the visceral afferent nerves are destroyed by pressure or anoxemia. Then pain decreases or almost ceases. By this time distention has caused complete occlusion of the capillaries and smaller veins and thrombosis develops. Impairment of the blood supply occurs first on the antimesenteric border where the vascular supply is poorest. When the reaction reaches the serosa so that pain of peritoneal origin is present rebound tenderness can be elicited and rigidity develops.

As more and more blood is pumped into the appendix the smaller vessels rupture and hemorrhage occurs. By this time the appendiceal wall distal to the point of obstruction is thinned by distention and the mucosa has become ulcerated and destroyed as a result of pressure necrosis. As necrosis occurs bacteria enter the tissues and fever, rapid pulse and leukocytosis furnish further evidence of the absorption of products of dead tissue. If the appendix is not able to overcome the obstructive mechanism perforation follows usually at one of the infarcted antimesenteric areas and the character of the pain is again altered due to the release of pressure. Westphal (cited by Bowers) suggested that because anaerobic gas forming bacteria are present in the lumen of the appendix the perforation is really an explosion which clinically it may resemble.

The experience of most observers is that the obstructive type of acute appendicitis is likely to exhibit a more severe and at the same time a more misleading symptom complex than the bacterial variety. The pain tends to come on suddenly to be colicky in character and to be extremely severe as compared with the more gradual onset and milder and more continuous character of the pain in nonobstructive appendicitis. Equally characteristic as well as highly misleading are the delayed appearance of temperature elevations and increased pulse rates, the unaltered leukocytosis and the usual absence in the early stages of localized tenderness. It is imperative to bear the picture of

obstructive acute appendicitis constantly in mind in the diagnosis of acute abdominal disease for the clinico-pathologic process just outlined makes clear the possible disastrous consequences of delay.

### DIAGNOSTIC METHODS AND OTHER CONSIDERATIONS OF DIAGNOSIS

In view of what has been said concerning symptoms physical findings and similar considerations in acute appendicitis not a great deal of time need be spent discussing diagnostic procedures. The diagnosis of the disease is entirely clinical. Other than a routine urinalysis and a total and differential white blood cell count and perhaps examination of a stool specimen for parasites laboratory measures are of little or no assistance. There are few diseases in short in which the physician and the surgeon must rely so completely on their clinical diagnostic ability.

**History.** The history must be taken with great care and in full detail in every suspected case of acute appendicitis. It should begin with a specific inquiry whether or not the appendix has already been removed. If the patient says that it has not been and if he bears no scars of abdominal surgery the statement may be taken at its face value. If he says that it has been and more particularly if a female patient says that it has been the inquiry should be pursued further particularly in a woman who has been submitted to pelvic surgery for in a small proportion of cases even a definite statement by the patient will prove to be incorrect (p. 126). It is extremely unfortunate that surgeons are not in the habit of supplying their patients after surgery with a written statement of the nature of the operation performed. It would be of great value in circumstances such as these.

The history should include the patient's age, occupation, the exact time and mode of onset of the attack, a specific inquiry concerning the last time he felt perfectly well, the duration of the illness, by the clock, the symptoms in elaborate detail and in the order of their appearance, the possible relation of the symptoms to menstruation, urination, defecation and the ingestion of food, including a specific inquiry about the last meal really enjoyed, the past history with particular reference to similar previous attacks and previous surgery. It is useful to inquire whether the patient particularly if he is a child has passed worms or has been treated for worms. All the details of the abdominal pain should be inquired into, including the onset, character, radiation, localization, relation to urination, defecation, ingestion of food, vomiting and similar considerations. The specific question should be asked whether a purgative has been taken, if it has been the patient



should be asked specifically whether it was vomited if so whether it was repeated and what its effect has been including possible alteration in the original symptoms. It is well to permit the patient to tell his own story so far as possible before making inquiry about special symptoms and other considerations but if the information is not secured specific though not leading questions should be asked.

*Physical Examination* Physical examination should be carried out in detail and should include (1) inspection of the general appearance, facies and posture, (2) inspection of the tongue (3) determination of the temperature, pulse, respiration and blood pressure (4) examination of the chest by inspection palpation percussion, and auscultation (5) examination of the abdomen by inspection palpation percussion and auscultation (6) examination of the hernial orifices (7) rectal examination in all patients (8) pelvic examination whenever it is not contraindicated by age or other reasons in all female patients (9) examination of the spine and demonstration of the knee jerks in all adult subjects. The reasons for the majority of these procedures are self evident.

*Examination of the Abdomen* Examination of the abdomen begins with inspection to determine the respiratory excursion the presence or absence of scars deviations from the normal contour caused by distention or masses and other gross variations from the normal.

Before palpation of the abdomen is begun it is important that the patient be made as comfortable as possible. Better relaxation is secured if two or more pillows are placed beneath the head and he may prefer to lie with the right thigh flexed on the abdomen during most of the examination. Asking him to breathe through his mouth also aids in relaxation. It is sometimes advisable, if examination cannot be carried out satisfactorily otherwise to give a nervous or hypersensitive patient a few whiffs of nitrous oxide or some other gaseous anesthetic so that muscular rigidity and the presence of inflammatory masses can be demonstrated or excluded when relaxation is complete. The preliminary use of hot packs is occasionally useful for the same purpose.

It is a wise plan when the patient has been made as comfortable as possible to follow the example of an astute old Scotch physician and say to him in effect "Put your finger where you be bad." The examiner then systematically palpates all areas of the abdomen beginning with the epigastrium and passing on to the umbilical area the left lower quadrant and other areas leaving the right lower quadrant (or whatever region the patient has indicated as the principal source of his distress) to the last. Palpation is begun gently to demonstrate super

ficial tenderness which is seldom present in acute appendicitis until parietal peritonitis has developed and is gradually deepened. When the rest of the abdomen has been thus examined the right lower quadrant is finally palpated preferably while the patient's attention is distracted by conversation or in some other manner. Pressure is gradually increased the faces meantime being carefully but unobtrusively watched. If tenderness is so great that a patient of normal self-control and normal nervous sensibilities cries out or cringes from the examining hand the diagnosis of acute appendicitis is established almost at once. A stoic patient however or a patient fearful of operation may deliberately control himself and may give no evidence of pressure pain or may actually deny its existence. A patient with a thick abdominal wall or one whose appendix is retrocecal or extraperitoneal or is covered with a thick fat omentum may also reveal no tenderness on palpation.

The area of maximum deep tenderness is identified by gradually increasing pressure of the hand over various areas of the right lower quadrant. Appendicitis may be present even if the maximum tenderness is not exactly localized but as a general rule if McBurney's point of tenderness can be demonstrated one is reasonably safe in excluding every other disease. In doubtful cases the examiner may perhaps prefer to attempt to identify some one of the other points of maximum tenderness which have been described (p. 129) but as has been indicated there are decided differences of opinion concerning the value of most of them.

Care must be taken in testing for abdominal tenderness to distinguish between the tenderness which the physician elicits by his maneuvers and the subjective pain of which the patient complains and which in the absence of proof to the contrary must be accepted at its face value.\* In difficult cases an attempt may be made to differentiate parietal from visceral pain by applying deep pressure to the area of

\* A recently observed case emphasized the possible origins of tenderness in acute appendicitis. A young man who was first seen a few hours after the onset of nausea and vomiting was thought to be suffering from gastro-enteritis but because acute appendicitis could not positively be excluded he was examined at hourly intervals for 6 hours by the attending surgeon and two residents. At the first 4 examinations no tenderness whatsoever was elicited. At the last 2 examinations the patient complained of tenderness but stated quite frankly that the complaint might be imaginary and might be explained by the fact that his attention had been directed to the possibility by repeated examinations and questions. It was also thought that too zealous examinations might be responsible for actual tenderness. That the complaint was real however was proved by the finding of an acutely inflamed appendix containing purulent fluid when operation was performed 9 hours after the onset of symptoms.

maximum tenderness while the patient elevates his fully extended legs. If the pain is relieved as he does so it is probably visceral in origin. If the pain is accentuated or if attempted elevation of the legs causes too much pain for the maneuver to be completed it is probably peritoneal in origin.

The presence or absence of muscular rigidity is also determined by deep pressure over the abdomen. Voluntary rigidity or guarding which is a purposeful muscular defense under the control of the patient must be distinguished from involuntary muscular rigidity which is not under such control and which indicates inflammatory involvement of the peritoneum. Spontaneous rigidity is sometimes increased by voluntary splinting of the muscles when the patient is instructed to take a deep breath.

Early in the illness muscular rigidity is often demonstrable only by deep pressure in the right iliac fossa or by comparison with the findings on the unaffected side. Cope has suggested if it is suspected that one is dealing with an individual with a hypersensitive nervous system which reacts readily to any sort of stimulus that an endeavor be made to elicit other reflex phenomena. If the same exaggerated response is elicited as when the rectus muscle contracts excessively on superficial palpation the same degree of response to internal stimulation can be assumed and the observation can be evaluated accordingly.

From what has been said it is clear that the absence, presence and degree of muscular rigidity must be interpreted with care. Clear cut muscular rigidity or even simple resistance in the right lower quadrant if associated with local tenderness almost always warrants a diagnosis of acute appendicitis. If the findings are slight or if rigidity is absent the interpretation is more difficult. There should never be any attempt however to correlate the type of pathologic process presumed to exist with the degree of muscular rigidity present. Simple inflammatory edema of the subperitoneal tissues is often associated with a marked degree and extensive disease may be present while the muscles are lax.

Hyperesthesia is variously determined. Some observers prefer to pinch the skin and subcutaneous tissues between the finger and thumb thus drawing them away from the deeper structures. Cope recommended the use of a pin with a sharp point held at an acute—and always the same—angle to the skin so that it does not scratch while it is drawn lightly above the surface toward the costal margin from Poupart's ligament. The strokes are made vertically from below upward since it is easier to feel an increase than a decrease in sensitivity.

The test is carried out on both sides in several areas beginning at the midline and light strokes are also made across the midline. The amount of pressure must be slight and uniform. The test should be repeated if the patient is uncertain in his responses. In Copes' opinion slight exaltation of sensitiveness is sufficient to warrant a diagnosis of acute appendicitis and it is not necessary to demonstrate the entire right iliac triangle described by Sherrin.

**Percussion.** Percussion of the abdomen is not likely to be of great usefulness to a physician who does not practice it regularly and who is not experienced in the recognition of normal and abnormal tones and of variations in tone quality, pitch and duration. It is carried out by tapping the abdominal wall with the finger tip or tips in the same systematic manner in which palpation is carried out, preferably while the patient breathes deeply so that the abdominal wall and the viscera are brought as close together as possible. The ear is held close to the area tested. Murphy recommended that percussion be carried out by the piano method which as the name implies consists of tapping the abdominal wall in rapid succession with all the fingers exactly as if one were playing the piano. A tympanic note is rather constantly present over the cecum in acute appendicitis. Otherwise this method reveals very little in an early case though it may be helpful in the exclusion of peritonitis and intestinal obstruction in obscure cases and in the demonstration of these conditions in advanced cases.

**Auscultation.** Auscultation of the abdomen is another method not likely to be of great value to the physician who does not practice it regularly. Its purpose is chiefly the confirmation or exclusion of such conditions as ileus or intestinal obstruction. In early cases of acute appendicitis it does not reveal much unless a purgative has been taken then peristalsis is sometimes increased. It may be carried out by applying the ear to the abdomen but is better practiced with the aid of the stethoscope which is moved systematically over all areas.

**Special Tests and Maneuvers.** Various special tests and maneuvers have been proposed as diagnostic aids in acute appendicitis but most of them are unnecessary (as well as of doubtful value) if the history is carefully taken and if the type of physical examination described is carried out. Any maneuver that requires pressure on the abdominal wall particularly if it causes back pressure into the appendix must be carried out very gently for perforation may follow the use of forcible methods (p. 388).

The demonstration of rebound tenderness (Blumberg's sign) is probably the most useful of the various suggested tests. Deepening

pressure is made with the fingers over the appendiceal area and is then abruptly released. If pain follows it may be assumed that the parietal peritoneum is irritated by the presence of an inflamed appendix.

Murphy recommended pressure over the left lower quadrant on the assumption that if acute appendicitis is present pressure upon the inflamed organ by the contents of the colon or by other abdominal organs will cause pain.

Cope advised the obturator test which is performed by internal rotation of the flexed thigh to the extreme limit the obturator internus thus being put on stretch. This muscle being covered by dense fascia is not readily irritated by pelvic inflammation but if it is put through its full range of movement hypogastric pain is caused when pelvic disease is present including pelvic disease of appendiceal origin. The test is never positive in thoracic disease and is therefore of differential diagnostic value especially when rectal examination is unsatisfactory.

Cope also recommended the psoas muscle test. The patient lies on the left side while the physician extends the right lower extremity to its full length and then abducts it. If pain is felt in the iliac fossa on flexion or extension of the thigh an inflammatory condition adjacent to the psoas muscle may be assumed. Hyperextension of the thigh may be necessary to elicit the sign but even slight degrees of irritation are usually revealed by this maneuver if the abdominal wall is relaxed. The test is less valuable if the abdomen is rigid.

Slom's maneuver may be useful in the patient with a heavy muscular abdomen in which tenderness is always difficult to elicit. The patient keeping the knee straight raises the right heel from the bed while the examiner places his left hand over the right lower quadrant of the abdomen and applies pressure to it with the fingers of the right hand. The psoas muscle is thus put on tension while the appendix is compressed between it and the left hand of the examiner.

Rovsing's sign (also known as Owen's test) is of special value in cases in which the colon is full of gas (fig 45). Firm pressure is made over the colon on the left side of the abdomen so that the gas in the bowel is forcibly pushed toward the cecum until pressure on an inflamed appendix forces the patient to complain of pain. Usually a considerable degree will be tolerated but if the pressure is maintained long enough to drive some of the gas out of the blind end of the large intestine the sudden removal of the hand permits the return of gas into the cecum and the increased pressure so distends the inflamed appendix that the patient may cry out with pain.

Altschuler's maneuver is intended to assist in the diagnosis of

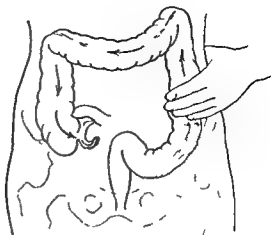


Fig 45 Retrograde inflation of cecum by pressure on descending colon (Rovsing's sign)

**retrocecal acute appendicitis** The third fourth and fifth fingers of the left hand are laid on the outer border of the right ilium while the left index finger is placed on the inner border. The index finger is then gradually moved downward over the soft parts from the anterior superior spine. In acute retrocecal appendicitis a circumscribed area of tenderness and muscle spasm usually small may be identified over the right pelvic margin and may be compared with the similarly located presumably insensitive area on the left side.

**Rectal and Pelvic Examination** Rectal examination is carried out with the patient on the back or the left side preferably on a hard table. Examination on a bed particularly if the mattress is soft yields considerably less information. The well lubricated rubber gloved fore finger of the right hand is introduced into the rectum for 3 or 4 inches and sweeps the pelvis in all directions while the left hand palpates the right iliac fossa. If the fingers of the right and left hand can be pressed toward each other without causing pain the appendix is not likely to be diseased unless it lies in such a position over the pelvic brim that it is missed entirely by the exploring fingers. The examination should be repeated within 2 or 3 hours in obscure cases. In early cases unless the appendix is low in the pelvic cavity examination per rectum may not reveal a great deal except in children in whom the appendix is more readily accessible to the examining finger. If peritonitis has developed bilateral or generalized tenderness will be elicited. In uncomplicated cases tenderness is usually confined to the right side.

The intelligent patient should be warned that a rectal examination

is necessarily a somewhat uncomfortable procedure and should be asked to distinguish between discomfort and actual distress. An excitable or unintelligent patient is not likely to respond to an appeal for co-operation. In older persons the discomfort of hemorrhoids, fissures and similar rectal conditions sometimes introduces a factor of confusion. In stout persons examination per rectum may not be helpful.

Yodice described a maneuver intended to differentiate muscular contraction from peritoneal reactions and peritoneal reactions from other causes. The patient lies in the dorsal decubitus with the feet resting on the bed and the thighs abducted and flexed to  $90^\circ$ . The index and middle fingers are introduced into the rectum and the left hand palpates the abdomen at the site of the muscular contraction. If the contraction does not disappear at the moment the anal sphincter is dilated it may be assumed that it is due to peritoneal irritation. If the contraction disappears the assumption is that the pain is of a colicky type for which emergency surgery is not required. The maneuver can be repeated several times until the interpretation is clear. Evidently according to Yodice dilatation of the anal sphincter produces spinal and sympathetic reflexes which modify muscular contraction of the abdominal wall. If however the reflex is intense as in peritoneal irritation dilatation of the sphincter is incapable of interrupting the reflex arc.

Pelvic examination is useful chiefly for differential diagnostic purposes to exclude gynecologic diseases. It is likely to be negative in early uncomplicated cases. In late cases a pelvic abscess can sometimes be diagnosed by the finding of a tender fluctuant mass pressing on the anterior rectal wall.

*Morphine.* Zierold proposed the use of morphine as a diagnostic method in acute abdominal diseases including acute appendicitis on the ground that abdominal muscle spasm has two components, a local spinal reflex and a widespread protective response to cortical stimulation. If the latter reflex could be eliminated as it can be by the use of morphine any spasm which might remain would be caused by an aggravated spinal reflex and the segment affected or even the site of irritation would thus be identified. The gross diagnostic error for Zierold's series of 150 cases was 10.3 per cent. No mistakes were made when the method was tested on 100 normal controls. There were 4 errors in the 44 cases of acute appendicitis. Most observers would not agree with the author's conclusion that the percentage of error by this method is no higher than observational error and would be likely to consider the use of morphine under such circumstances as highly dangerous.

*Peritoneal Aspiration.* The various methods of peritoneal aspiration

proposed for the diagnosis of acute appendicitis are obviously useful only when the pathologic process has reached the serosal coat of the appendix and invaded the peritoneal cavity. Touroff, who stated that peritoneal aspiration had been used as a diagnostic method with great satisfaction at Mount Sinai Hospital in New York ever since it was first proposed by Solomon in 1906, described the technique as follows:

An 18 gauge lumbar puncture needle with stylet in place is introduced into a wheal produced by novocaine just below the level of the umbilicus either immediately lateral to the right rectus muscle or just over it. The needle is advanced in a slightly cephalad direction so as to traverse the parietal peritoneum with the bevel still facing downward. The stylet is then withdrawn and an accurately fitting 10 cc syringe is attached to the needle which is moved in various directions while suction is applied. Even a single drop of fluid can be examined microscopically and culture can be carried out if a sufficient amount is available.

The findings are interpreted as follows. Fluid containing only polymorphonuclear leukocytes is regarded as compatible with the diagnosis of acute appendicitis and the presence of an early peritoneal reactive process though a negative result does not exclude the diagnosis. The presence of bacteria establishes the diagnosis of peritonitis and aspiration to the left side of the abdomen will permit the determination of whether the involvement is generalized or localized.

Meleney, in discussing Steinberg's contribution, also advocated examination of the peritoneal fluid in doubtful cases before operation. The risk, in his opinion, consists not in possible damage to the intestines or blood vessels, the danger of which he considered minimal but in the possibility of obtaining a specimen of fluid which is not representative and therefore of delaying an operative procedure which should be carried out. In his personal experience widely different results could be obtained in the same case depending upon how near to the focus of infection the specimen had been secured. Steinberg and Martin (p. 73) believe that this danger does not exist.

#### THE CLINICAL SYNDROME IN RELATION TO THE ANATOMIC POSITION OF THE APPENDIX

There is undoubtedly a frequent correlation between the position of the appendix and the clinical picture. Acute appendicitis may present though I do not myself believe that it is either necessary or altogether wise to stress it unduly. There is not always complete agreement on the



part of various observers on the symptoms and findings likely to be associated with special positions of the appendix and my own experience is that too much hair splitting can lead only to grief.

The details that follow should therefore be accepted with reservations.

*The Ascending Appendix* When the appendix lies in the iliac fossa the physical findings are those of the so called typical case that is tenderness and rigidity. Because of the close relation of the appendix to the abdominal wall it is usually fairly easy to determine whether rupture has occurred whether the peritoneal reaction is limited or diffuse or whether abscess formation has taken place. On the other hand the mass felt by the examiner which he so confidently believes to be an abscess frequently proves to be an omentum wrapped up appendix. The differentiation is extremely important for advocates of expectant treatment would delay surgery in such a case and might thereby permit irreparable damage to occur.

*The Retrocecal Appendix* There is considerably less agreement about the picture of the retrocecal appendix. According to Cope the initial pain is frequently in the right lower quadrant and is usually mild vomiting is less frequent and muscular rigidity is often much less than would be expected by the stage of pathologic change found at operation. Reid was of the same opinion adding that the pain was frequently so mild that the patient might not seek medical advice until rupture and abscess formation had occurred though rupture into the general peritoneal cavity might be forestalled by the buried and protected position of the appendix. He described the point of maximum tenderness in retrocecal appendicitis as somewhat above McBurney's point. In his experience fever was likely to be lower and leukocytosis somewhat higher than usual. Altschuler in contrast to the observers just quoted described the pain of acute retrocecal appendicitis as likely to be generalized and to begin in the region of the stomach. Muscle spasm in his experience was likely to be absent and the only positive finding might be slight tenderness over McBurney's point.

Collins found the most frequent sign of retrocecal appendicitis to be the production of intense pain by deep steady pressure on the right flank lateral to the right psoas muscle. In a series of 751 cases of this type of disease the so called classical picture was present in only 142 cases the remainder presented the same bewildering variety of symptoms and signs likely to be found in any other large series of cases. In 218 cases rectal examination furnished the only positive signs. The diagnosis of acute appendicitis was made correctly in 554 of the

751 cases but in only 304 was the retrocecal position diagnosed which means that the position was missed in approximately three fifths of all cases

*The Pelvic Appendix* There is no more general agreement about the clinical picture produced by disease in a pelvic appendix than there is about the picture produced by disease in a retrocecal appendix. The pelvic type however is extremely important because an appendix in this location may be on the point of rupture into the general peritoneal cavity while there is still little or no warning of the impending catastrophe and still more important while not a single adhesion exists to limit the infection. This possibility is not emphasized as it should be in most textbook discussions of acute appendicitis and the results of the failure are clear from such a report as that by Brunn who stated that 90 per cent of the ruptured cases of acute appendicitis he saw in consultation were of the pelvic type

According to Cope the early symptoms of acute disease in a pelvic appendix do not differ from those produced by an appendix lying above the pelvic brim except that rigidity is not present in the early stages epigastric pain may last longer and there may be bilateral pain in the iliac fossae. Persistence of the epigastric pain is due to distention of the appendix and the resulting severe peristaltic contractions. Iliac pain can usually be elicited by deep pressure at the brim of the true pelvis but to localize it in the right lower quadrant is not always possible. When rupture occurs epigastric pain decreases. Pelvic peritonitis which may occur either on the right side or in the depths of the pelvis is less painful than other varieties of peritonitis examination is usually negative because rigidity of the abdominal muscles is absent and the patient actually seems better. Within 3 or 4 days either an abscess forms which is the most favorable outcome or the pelvic peritonitis extends upward and the usual findings become apparent

According to Brunn pain is the most constant symptom of pelvic appendicitis. It begins in the epigastrium or around the umbilicus but soon settles in the lower abdomen. Localization is more frequent on the left side or on both sides than on the right side is a finding which should in itself arouse suspicion. Thereafter Brunn's description of the clinical picture of pelvic appendicitis differs even more considerably from Cope's. If rupture occurs the patient in his experience suddenly becomes acutely ill. There is abrupt severe pain marked generalized rigidity and dullness over the right or left side of the abdomen or over both sides and sometimes over the pubic region also. The pain is usually limited to the infraumbilical region but may occur in the

epigastrium and the location especially if shock occurs, as it occasionally does may suggest a diagnosis of ruptured peptic ulcer

Reid's description differs from those of Cope and of Brunn. In his experience the pain of pelvic appendicitis is often no more than discomfort. It may be on the left side or bilateral. Often there is no vomiting and sometimes nausea is also absent. Examination reveals generalized tenderness more marked in the lower abdomen. Muscle spasm is frequently absent or is so mild that it is not demonstrable if it is present. The point of maximum tenderness may be in the midline between the umbilicus and symphysis or on the left side.

However much observers may disagree in their descriptions of most aspects of pelvic appendicitis they are agreed on at least two points: the frequency of urinary and rectal symptoms because of the relation of the pelvic appendix and bladder and the importance of pelvic examination. Diarrhea with or without tenesmus may be present and the patient may have a constant desire to move the bowels. Sometimes the clinical picture is so mild that other symptoms are overshadowed by the diarrhea and the physician may not make a complete examination until perforation occurs and severer symptoms are precipitated.

Questions about the patient's urinary function are of great importance in the pelvic variety of acute appendicitis. Urinary symptoms are likely to be particularly pronounced in children because the bladder is higher in the abdomen at this age. Often dysuria, frequency and hematuria are so pronounced that all the attention is centered on the possible bladder and renal factors (p. 118) and cystoscopy and similar examinations are carried out.

In many cases of pelvic appendicitis rectal examination furnishes the clue to diagnosis. Right-sided tenderness may be elicited by pressure of the examining finger or a definite tender swelling can be made out which in early cases is the pelvic appendix and in advanced cases is a pelvic abscess. If however the appendix is situated so high in the right posterior quadrant of the pelvis that it is impossible to palpate it diagnosis must be made on the clinical findings and the absence of more usual signs and symptoms.

**Conclusions.** Obviously authorities do not always agree on the clinical picture of acute appendicitis in relation to the location of the appendix, and reliance on one description or another may be very misleading. Little more than the following general statements seem warranted:

- 1 Rigidity and localizing signs are usually present when the appendix is in contact with demonstrable areas of parietal peritoneum
- 2 Rigidity is likely to be absent at least before perforation occurs if the appendix is not in contact with a demonstrable area of parietal peritoneum that is if it lies in the depths of the pelvis behind the ileum or in front of the sacrum with coils of intestine lying in front of it
- 3 Rigidity may be present in the right lumbar region if the appendix lies posteriorly or lies far forward anteriorly
- 4 Rigidity may be present in the upper abdominal muscles if the appendix points toward the liver
- 5 Rigidity is likely to be absent if the appendix lies in the pelvis Deep palpation low in the iliac fossa may elicit tenderness which is frequently bilateral and rigidity is sometimes observed over the attachment of the rectus muscle to the pubic bone

#### DIAGNOSIS IN RELATION TO THE PATHOLOGIC PROCESS

On the surface it seems logical to attempt to relate the clinical picture in acute appendicitis to the pathologic process. One would scarcely expect the same clinical picture in a patient with simple acute appendicitis as in a patient with spreading peritonitis. On the other hand logic to the contrary patients with conditions as widely apart as these frequently present precisely the same clinical symptoms. Within a period of 8 hours on the same ward I once saw 3 patients who all presented the classical picture of acute appendicitis. The first had acute mesenteric lymphadenitis. The second had a gangrenous appendix with a minute perforation. The third had an appendiceal abscess. In 7 cases described by Sauer and Bailey in which the clinical picture was substantially identical the appendix was negative in 1 case while in the remaining 6 cases the pathologic processes included simple acute disease, gross and microscopic gangrene, perforation and abscess formation.

As a matter of fact it is extremely dangerous for the physician to dogmatize about the stage of the pathologic process. It is naturally gratifying to make an exact pathologic diagnosis but if one follows the principle of immediate operation in acute appendicitis the attempt is frequently not worth what it costs on the principle succinctly stated by Deaver that a hair splitting diagnosis often gets a patient nowhere except to the grave. John B. Murphy in spite of the large percentage of cases in which he predicted exactly the conditions which would be found within the abdomen gave up the plan because of some rude

awakenings and thereafter contented himself merely with determining that the patient had acute appendicitis.

It is equally unwise to attempt to diagnose the stage of acute appendicitis on the basis of elapsed time since the onset. The duration of any acute abdominal disease is naturally important from many points of view. But an inherently mild case of acute appendicitis may still be a mild case and perhaps on its way to complete resolution at the end of 24 or 48 hours after the onset or even longer while another case particularly of the obstructive type may present gangrene or rupture 11 or 8 hours after the onset or even earlier. Moreover to permit time to pass in order to observe possible developments in a given case is a policy which can lead only to disaster. There should positively be a time limit for making the diagnosis in a suspected case of acute appendicitis and I myself would set it at not longer than 6 hours. During this period the physician if he cannot remain with the patient continuously should see him at least every 2 hours and preferably oftener. A still safer plan for the physician who suspects that he may be dealing with acute appendicitis is to call a surgeon into consultation at once so that the responsibility of diagnosis shall be jointly borne and that the patient may profit from the wisdom and experience of two observers.

*The Lucid Interval (Period of Dangerous Calm)* Although attempts to diagnose the stage of the pathologic process in acute appendicitis are unwise it is nonetheless sometimes possible to determine about what has occurred. This is frequently though not always true when definite peritonitis which is a complication and not an intrinsic part of the disease has developed. It is frequently possible when there has ensued what Bower has called the *lucid interval* and what Dieulafoy originally called the *dangerous period of calm*. This particular stage has such important implications that Bower's description of it should be quoted in some detail. In his own experience over a 3 year period cases from this group furnished 15 per cent of all deaths from appendicitis.

The lucid interval is characterized by a subsidence of symptoms and in absence of physical signs frequently interpreted as indicative of resolution of the acute disease. Actually the apparent retrogression is due to partial or complete gangrene of the appendix. In both resolution and gangrene the subsidence of symptoms and signs is due to diminished intra-appendiceal pressure. In gangrene it is additionally due to devitalization of tissue. The appendix which is dead wrote Murphy like the patient who is dead is incapable of making its voice heard.

The typical history in such cases according to Bower includes colic and localized tenderness in the right lower quadrant as the

result of increased intraluminal pressure. Then all signs and symptoms disappear with the possible exception of a moderate temperature elevation because gangrene has occurred and nerve terminals are devitalized. The greater the amount of tissue involved in the gangrenous process the more complete is the period of lucidity. If operation is done at once life may be saved. If it is not or if conservative treatment is deliberately employed an abscess may or may not develop. If however the patient is assumed to be recovering and is permitted to resume his usual activities or if he takes food or fluid by mouth perforation may occur at once. Then there is no doubt of what has happened. The way to forestall the tragedy Bower concluded is to be certain that a patient whose disease is supposed to be subsiding is kept under observation with food and fluids withheld until it is evident that subsidence of the disease process and not the development of gangrene is the cause of his apparent well being.

### PRINCIPLES OF DIAGNOSIS

The first principle of correct diagnosis in acute appendicitis is the realization that it is the commonest of all surgical diseases and that if only by the operation of the laws of chance it is more likely to be the cause of acute abdominal symptoms than is any other disease. Deaver when he listed acute abdominal conditions in the order of intensity put acute pancreatitis first and acute appendicitis last. When he listed them in the order of frequency he headed the list with acute appendicitis and demanded that the physician confronted with an apparent abdominal emergency should consider that disease first last and all the time.

There is almost no conceivable circumstance under which Deaver's warning should not be remembered. Acute appendicitis is a disease which spares no race social station or sex. It spares no age for it may occur at any period of life from the cradle or more correctly the uterus (p. 341) to the grave. It may occur while the victim is awake or asleep at work at play in the moving pictures in church on land on sea and in the air in the center of civilization in the heart of the wilderness on the battlefield and in the physician's office where at least two persons of my acquaintance developed their symptoms while awaiting consultation for other complaints.

• The second principle of diagnosis is the realization that acute appendicitis may be initiated with or attended by almost any conceivable

symptom or sign regardless of how bizarre and unrelated it may be. As a result the diagnostic possibilities are almost unlimited. Even the description of a classical case of the disease must be qualified somewhat as follows. Acute appendicitis begins with abdominal pain which may appear first in the epigastrium umbilical region pelvic region or elsewhere in the abdomen. The pain may or may not later localize in the right lower quadrant. It may be accompanied with or followed by anorexia, nausea, and/or vomiting. It may be accompanied by constipation or diarrhea or the bowel function may remain normal. It may or may not be accompanied by urinary symptoms. The temperature may or may not be elevated. The pulse rate is usually but not always faster than normal. Local tenderness can usually be elicited on abdominal examination and sometimes by rectal examination. Local rigidity and superficial hyperesthesia may or may not be present. These symptoms and signs may be mild or severe, may appear in variable order and may be accompanied with or preceded by a wide variety of other symptoms and signs.

Generalizations any more precise than these are likely to be incorrect. It is not true as Murphy stated that if fever precedes pain or if vomiting precedes or accompanies the first bout of pain or if fever is not present at all, acute appendicitis can usually be put out of mind. It is not true as Moynihan stated that if pain be not the first symptom then the disease is not acute appendicitis. It is not true as many believe that pain which begins in the right lower quadrant is not acute appendicitis though the converse is true, that pain which originates anywhere in the abdomen must always be regarded as acute appendicitis until it is proved not to be. It is not true, as some observers state, that identification of a point of maximum tenderness is necessary for a diagnosis of acute appendicitis. Precise localization is not likely to occur if the appendix is pelvic or retrocecal or if it lies in a fossa on the posterior peritoneal wall. On the other hand if McBurney's point can be demonstrated one is reasonably safe in eliminating every other disease. That even this statement must be qualified however is evident in such a report as that by Altemeier and Holzer on primary torsion of the omentum in 5 of the 6 cases tenderness was present at McBurney's point and they regard the clinical picture in practically all cases as essentially indistinguishable from acute appendicitis.

More and more as their experience with this disease increases physicians are coming to accept Wangensteen's point of view that if abdominal pain and local tenderness are present and if they cannot

reasonably and definitely be explained as due to another cause the physician is justified in making a diagnosis of acute appendicitis and is indeed obligated to make it if only because he cannot prove that that is not the diagnosis. I share that conviction and I believe that to its growing acceptance and not to chemotherapy, antibiotics or any similar consideration is to be attributed the improved survival rate observed in recent years in acute appendicitis.

*Diagnosis by Exclusion* One of the difficulties in the diagnosis of acute appendicitis is the need for haste which the urgency of the disease always demands. Painstaking investigations which would be safe and desirable under other circumstances are dangerous and wrong in this condition. The time factor is the most important of all considerations. Therefore the physician bearing in mind the overlapping nature of the symptom complex in most acute abdominal diseases must arrive at the diagnosis of this special state on the basis of his own past experience, the findings in the particular case and the exclusion of other diseases, particularly nonsurgical diseases.

Diagnosis by the law of averages as has been intimated is useful in suspected acute appendicitis provided that the proper reservations are made. An acute abdominal condition has more chance of being appendicitis than anything else. The disease is relatively infrequent in the aged subject though it cannot be positively excluded merely on that account. If symptoms come on in the early morning hours waking a person previously in good health from sound sleep acute appendicitis is the first diagnosis to be considered. A patient who lies with his right leg drawn up on his abdomen is likely to have acute appendicitis. A patient with acute appendicitis does not usually move about the bed nor does he usually lie rigid and immobile. A patient with a history of previous attacks is likely to have acute appendicitis during another similar acute illness though the absence of such a history is of no diagnostic value at all.

If the diagnosis cannot be arrived at by any other means exclusion although mechanical offers a very practical method. A definite mental rehearsal of all possible conditions including the most unusual and most unlikely sometimes clears up an obscure diagnosis which more scientific methods have failed to clarify. Diagnosis by exclusion is based on the obvious consideration that before one can diagnose or eliminate a given disease one must remember that it may be present. In acute appendicitis this means literally that the only patient who may not have the disease is the person whose appendix has already been removed.



## CONCLUSIONS

The reader who has finished the perusal of this particular chapter may complain that he is left with a very confused picture of acute appendicitis. The statement would be correct. It should not, however, be made as a complaint. The syndrome of acute appendicitis is confused in from 25 per cent of all cases by generous estimates to 50 per cent by less generous—and probably safer—estimates. That is why it has been pointed out elsewhere (p. 402) the description of acute appendicitis in many textbooks and systems is open to criticism. That is why the mortality of acute appendicitis still remains at an unnecessarily high level.

In other words the classical triad of abdominal pain, nausea and vomiting and right sided tenderness and rigidity is not the real syndrome of acute appendicitis in a third or more of all cases. The real syndrome in that group of cases is a widely variable syndrome—a confused and disorderly syndrome in which almost any symptoms and signs may appear in almost any sequence. The physician who bears that fact in mind is likely to make the diagnosis of acute appendicitis correctly and promptly and to have a higher percentage of accuracy than the physician who pins his faith to the less confusing but thoroughly incorrect concept of the so called typical picture. Only by emphasis on the atypical case of acute appendicitis can lives be saved in this disease.

## (VIII)

### *The Differential Diagnosis of Acute Appendicitis*

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#### GENERAL CONSIDERATIONS

Elmson has suggested a sound plan of procedure in the diagnosis of a possible acute surgical condition. The surgeon should determine (1) whether the symptoms and signs are caused by a medical or a surgical state (2) whether the condition is one which demands emergency measures (3) the exact diagnosis if it can be made. If however a decision can be made concerning the first two points the third from both the physician's and the patient's standpoint is considerably less important.

Elman who suggested somewhat the same plan of procedure in possible acute appendicitis was more specific. (1) The physician must exclude medical diseases that may produce acute abdominal pain such as coronary disease, primary peritonitis, amebic dysentery, and typhoid fever. In this group of conditions operation would be harmful and could be fatal. (2) He must exclude other nonsurgical diseases such as spastic colitis and acute pelvic inflammatory disease. In this group of conditions operation would be a mistake but would be unlikely to have serious consequences. (3) He must exclude surgical diseases not related to the appendix such as perforated peptic ulcer and perforated Meckel's diverticulum. In this group of conditions surgery is necessary and no harm would be done by performing laparotomy on a mistaken diagnosis provided that an incision were made that would enable the surgeon to recognize and deal with whatever acute lesions were present.

Not to operate in acute appendicitis, Elman pointed out, is the really serious error. To operate and find a normal appendix and no other lesion needing surgical attention is a good mistake though it must not be made too frequently and medical diseases must have been carefully excluded before operation is undertaken. The physician who puts the burden of his diagnostic endeavors on the exclusion of conditions not connected with the appendix, particularly medical conditions, is entirely

justified in recommending operation in any given case in which he can not say positively that the patient does not have acute appendicitis. The surgeon who has operated under such circumstances and who finds a normal appendix should feel no embarrassment and should not be censured. Furthermore as Moynihan once said exploration for the purposes of surgical inquiry carries practically no risk today though that is naturally no justification for the reckless use of laparotomy as a means of diagnosis.

It must always be remembered that surgery implies trauma. A desperately ill patient ill with a surgical disease that only operation can remedy must take his chances with the undesirable effects of operation. A desperately ill patient ill with an illness that cannot be clearly classified as either medical or surgical and that may be a condition remediable only by surgical measures must also take his chances with operation though the possible consequences must be realized and every reasonable diagnostic method must first have been employed. When a situation arises in which surgical diseases are mimicked by purely medical states and differentiation proves impossible then the patient's chances are better with surgical exploration than without it and more lives are lost by withholding exploration under such circumstances than by carrying it out. The mortality that attends exploratory laparotomy even in a seriously ill patient is far less than the mortality that attends the nonsurgical treatment of acute appendicitis.

There is general agreement about the wisdom of and the justification for this course of action. In no other field of surgery wrote Heyd are errors of omission attended with such frightful mortality and errors of commission attended with such slight risks. Mitas in a discussion of Gieritz's contribution on peritonitis spoke even more strongly. But while I am unconditionally opposed to any and all operations performed without a reasonable foundation I would rather see a barrel full of normal appendices removed through honest error than see a single avoidable death caused by neglect of timely surgical intervention.

It would be impossible in a book of this length to go into detail concerning the differential diagnosis of all the diseases with which acute appendicitis may be confused. The best that can be done is to point out the possibilities of diagnostic confusion to illustrate certain of the more important sources of confusion and to summarize briefly the most important diagnostic considerations in the most important medical and surgical states with which acute appendicitis may be confused.

Fitz was perfectly aware of the diagnostic difficulties that may arise in acute appendicitis. In addition to the historical confusion with in

inflammation of the cecum which he himself cleared away he also discussed crises of the spine and hip suppurative nephritis intestinal tuberculosis cancer of the cecum intestinal obstruction from intussusception and strangulation biliary colic renal stone and gynecologic conditions

### THE DIAGNOSIS OF ACUTE APENDICITIS COINCIDENT WITH OTHER DISEASES AND CONDITIONS

The warning is necessary that acute appendicitis like any other disease may develop coincident with a pre existing disease. A tabulation of the other diseases present when acute appendicitis developed and of the circumstances under which it developed in the cases studied at the New Orleans Charity Hospital makes this point strikingly clear (Tables 3-4). Undoubtedly if the histories had been taken more carefully the list would have been more impressive. As it stands it is impressive enough for the associated mortalities show very clearly the confusion that may arise when acute appendicitis develops under unexpected circumstances.

Attention has been called elsewhere (p. 51) to the 166 cases 17 of which ended fatally in the surgical series at Charity Hospital and to the 13 cases in the 153 fatal nonsurgical cases in which the initial symptoms of acute appendicitis followed dietary indiscretions and were assumed for a time to be of dietary origin. Shrimp crabs oysters potted meat and similar articles of food are readily incriminated in gastro-intestinal disturbances. When young children gorge themselves on pecans and peanuts or eat twenty apricots or a dozen camphor balls a physician can scarcely be blamed for making a diagnosis of gastro-enteritis before he considers any other disease though the mortality of more than 10 per cent in this group of cases at Charity Hospital is proof of the disastrous consequences of the misinterpretation of events.

T. G. Miller reported a particularly striking instance of this kind.

A 40 year old man and his son both developed abdominal pain after eating oysters. The son took an enema and recovered promptly. The father continued to have diarrhea and generalized abdominal pain he became quite distended but had no nausea or vomiting. Abdominal tenderness was marked in the left lower quadrant and rectal tenderness was extreme in the midline. Operation 48 hours after the onset of symptoms revealed an acutely inflamed appendix with the tip in the pelvis and free fluid in the peritoneal cavity.

The observations made at the New Orleans Charity Hospital in respect to the development of acute appendicitis with other diseases

TABLE 3

CIRCUMSTANCES OF ONSET IN 6411 SURGICAL CASES OF ACUTE APPENDICITIS

	<i>Cases</i>	<i>Deaths</i>
Purgation	51	5
Menstruation	17	3
Coitus	1	1
Tonillectomy	2	
Mastoidectomy	1	
Extraction of teeth	2	
Vaccination	1	
Pregnancy	23	2
Abortion	3	
Puerperium	5	
Defecation	5	2
Urination	1	
During sleep	560	
Postprandial	85	1
Hospitalization for other causes	3	1
Dietary origin	166	17
Drinking spree	9	

\* The list is not intended to be complete merely typical

and conditions are not at all unusual Lesser and Kaufman for instance reported a case of gangrenous appendicitis associated with intussusception of the jejunum in a young boy whose illness developed while he was in a full plaster spica for fractures of the left femur and right tibia Lesser and Keshun reported a case of appendiceal abscess which involved an undescended testicle Jennings and his associates reported that in 65 cases 38 per cent of their total series acute appendicitis was preceded by or was coincident with upper respiratory infections every appendix in this group was gangrenous Taube reported a case of acute appendicitis associated with acute ulcerative colitis and cited two similar cases reported by Bockus in both of which the appendix was perforated and diagnosis was made only at necropsy

Easton reported a case in which appendectomy was performed in the course of Vincent's angina in the belief that the appendix had already perforated actually it was of the so called chronic variety In a similar case reported by Reuter and cited by Easton on the other hand the appendix showed beginning gangrene and the contents were purulent Easton noted ruefully that in his personal case Murphy's classic triad (pain nausea and vomiting and right sided tenderness) was present in its entirety and immediate appendectomy seemed urgently indicated

Rose reported the case of a 10 year old girl with mild symptoms of acute appendicitis who several hours after the onset of these symptoms received extensive second and third degree burns of the abdomen

TABLE 1  
COINCIDENCE OF OTHER DISEASES IN 641 SURGICAL CASES OF ACUTE APENDICITIS

	To 12 yrs Cases	Deaths	13-39 yrs Cases	Deaths	Over 39 yrs Cases	Deaths	Total Cases	Total Deaths
Onset with								
Upper respiratory infections	101	11	251	5	22	3	181	19
Pneumonia	2		2				9	
Measles			2		1		3	1
Typhoid			1				1	
Chickenpox	1						1	
Thyroid disease			2				1	
Whooping cough	1						1	
Rheumatic fever	1						1	
Menstritis							1	
Malaria	2		1		1		3	
Althia	1		2		1		10	1
Coccidiosis	6		5		1		1	
Pharyngitis			1					
Biliary tract disease			3		1		2	
Intestinal parasites			16		11		62	10
Cardiac disease	37	5	11		30		19	
Tuberculosis	1	2			1		2	
Urinary tract disease	3		28		17		30	4
Diphtheria					6		8	1
Brain tumor			1	1	1		1	
Carcinoma of lung	1						1	
Mumps							1	
Peptic Ulcer			11		6		21	
Mesenteric lymphadenitis	60		9				89	

thighs buttocks and other areas She was admitted to the hospital in shock Eight hours later the symptoms referable to the appendix increased in severity and operation was carried out through a McBurney incision made in the crust of a third degree burn treated with tannic acid The child recovered

*Acute Appendicitis and Hernia* The whole subject of the presence of the appendix in a hernial sac (inaccurately but generally termed hernia of the vermiform appendix) was reviewed in 1937 by Ryan who added to the 512 cases collected by Watson in 1923 from the literature 15 cases reported in the interim The total number of reported cases undoubtedly represents only a small proportion of the actual incidence since most writers on the subject have recorded relatively large numbers of personally observed cases Wakeley for instance among 2000 operations for hernia performed over a 22 year period found the appendix in a hernial sac 16 times his total personal experience thus including more cases than were reported in the literature from all sources over a 14 year period

The majority of reported cases of appendix in a hernial sac are not instances of acute appendicitis though there are a number of reasons why acute appendicitis should be fairly frequent under these circumstances In an inguinal hernia according to Watson the exposed position of the appendix and the contractions of the abdominal muscles may be responsible for considerable irritation and trauma In a femoral hernia acute disease could be excited by the narrowness of the hernial opening the pressure exerted by the sharp edge of Gimbernat's ligament and the trauma caused by active movements of the thigh The risk of strangulation of the appendix is increased when the meso appendix is thick and contains globules of fat Nearly all the recorded cases of serious strangulation occurred in femoral hernias Most recorded cases occurred in right sided hernias

Strangulated internal hernia may simulate acute appendicitis and acute appendicitis may form part of the pathologic process in this type of hernia Shrager and Riggs who recorded a case of strangulated internal hernia in which the clinical course was typical of appendicitis but the appendix was grossly normal called attention to the case recorded by Zoepfel in which the entire small intestine was incarcerated in the retrocecal recess and the appendix was perforated They also noted that the diagnosis is not usually made before operation in this condition and that the most frequent erroneous diagnosis is acute appendicitis

The appendix may be present in any type of hernia In Watson's



Fig 46 Appendix cecum and ileum visualized in sac of scrotal hernia after barium enema (from the University of Minnesota Hospitals Minneapolis)

collection of 512 cases it was found in an inguinal hernia in 55 per cent in a femoral hernia in 40 per cent in an umbilical hernia in 4 per cent and in other varieties in 1 per cent of the series Wakeley was of the opinion that the appendix is most likely to be found in an inguinal hernia in the male with visceroptosis the predisposing cause and in a femoral hernia in the female with repeated pregnancies the predisposing cause. The collected series show an age range from 6 weeks (this case occurred in a child born 2 months prematurely) to 79 years. Most instances occurred in persons past middle life and other abdominal contents such as the cecum the omentum or the small intestine (fig 46) were frequently also present in the sac. If the cecum was abnormally mobile or the meso appendix unusually long the whole appendix frequently was in the sac more often, not more than half to two thirds of the length was within the sac because adhesions or a mobile cecum or a short meso appendix held the organ within the abdomen.



Symptoms in the reported cases of acute appendicitis associated with hernia were chiefly pain and nausea and vomiting. Pain originated in the epigastrium or about the umbilicus as in the typical case of acute appendicitis and was often out of proportion to the size of the hernia. Pain associated with strangulation was likely to be dull, steady, and progressive. Abdominal tenderness was the rule but rigidity was likely to be absent because the infectious process was confined to the hernial sac and was therefore extraperitoneal. Fever might or might not be present. Physical findings included redness of the skin and edema and induration of the tissues overlying the appendix; the sac showed a tendency to increase in size as the illness progressed. When abscess formation had occurred fluctuation could be elicited.

Symptoms referable to the appendix were present in only 124 of the 512 cases collected by Watson, which explains why diagnosis was so frequently missed before operation. The high death rate in the series can obviously be explained by delay in diagnosis. Attempts to reduce the hernia by taxis add to the seriousness of the patient's condition when acute appendicitis occurs in a hernial sac and prompt appendectomy through the sac if that is practical is the method of procedure. In the past the majority of observers expressed the opinion that repair of the hernia should be deferred until a second operation. Modern chemotherapy and antibiotic therapy now permit the combined operations unless the appendiceal disease is advanced.

**Conclusions** The impressive fact about the coincidence of acute appendicitis and other diseases is not so much the frequency of such an association as the frequency with which the appendiceal disease is overlooked as indicated by the advanced pathologic process presented at operation in so many of these cases. The diagnosis is admittedly often puzzling under these circumstances and no rules can be laid down except as has been emphasized the warning to bear constantly in mind that acute appendicitis is a possibility in the course of any other disease.

#### THE DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS AND NONSURGICAL CONDITIONS

The differentiation of medical and surgical states is more than an academic problem in the diagnosis of acute appendicitis. Comroe in a paper on the nonsurgical causes of acute abdominal symptoms listed 9 groups and 27 subgroups of conditions to be differentiated and pointed out that the list was not inclusive and did not contain such conditions

as biliary colic or renal calculus for which later surgical intervention is usually necessary

Finney Sr listed 40 conditions which in the course of his practice he had been called upon to distinguish in their initial stages from acute appendicitis or vice versa including grippe measles tonsillitis scarlet fever smallpox rheumatism tabes dorsalis Henoch's purpura angio neurotic edema allergic manifestations (food poisonings) actinomycosis ruptured aneurysm of the renal artery hip disease osteomyelitis psoriasis abscess inguinal adenitis retroperitoneal tuberculous lymph nodes herpes zoster subcostal neuritis angina pectoris coronary thrombosis rupture of the left ventricle acute epididymitis in a right undescended testicle perinephritic abscess pneumonia diaphragmatic pleurisy and septicemia with multiple abscesses In 3 cases with almost precisely the same clinical syndrome Finney performed an appendectomy in the first only to have measles develop within 24 hours he refrained from operation in the second which also proved to be measles and he attempted to refrain from it in the third case because he thought it too was a case of measles only to be obliged to operate for a ruptured appendix a few hours later

*Respiratory Disease* The chest should be routinely and thoroughly examined in every patient with acute abdominal symptoms as part of the diagnostic procedure On the surface the history of a cold bronchitis chills fever cough and other evidences of upper respiratory infection point to a diagnosis of thoracic disease but they do not exclude acute appendicitis which may also be present and which may be the primary disease Moreover as has been pointed out (p 46) there is a fairly frequent though still unexplained association between acute appendicitis and upper respiratory infections

The patient with thoracic disease does not usually look like the patient with intraperitoneal disease The cheeks are flushed and the eyes are bright Often there are blisters on the lips or about the nose The movement of the alae nasi is likely to be accentuated and this sign should always be noted for it may precede other signs of pleurisy or pneumonia These signs are not evident when only acute appendicitis is present

The patient with pleurisy or pneumonia is likely to choose the lateral decubitus for comfort The patient with an intraperitoneal lesion is likely to lie on his back with one or both legs drawn up

In most acute diseases of the chest there is an elevation of temperature of  $2^{\circ}$  or  $3^{\circ}$  at the onset especially in young children Most acute abdominal conditions are not accompanied by any fever at all at the

onset and with only mild elevations for some time afterward. The absence of fever in older persons must not at once be accepted at its face value because of the delayed response of the bodily defenses in older age groups. The temperature in children with appendicitis is proportionately much higher than in adults and may resemble the level of the temperature in thoracic diseases. The pulse rate is usually more rapid in acute respiratory infections than in abdominal disease.

The respiratory rate is more likely to be increased in thoracic than in intraperitoneal disease, though voluntary or involuntary abdominal rigidity may be accompanied by a compensatory increase in the respiratory rate. The ratio between the pulse and the respiration is often of great diagnostic value. In pneumonia the normal 4:1 ratio is considerably reduced and a ratio of 2:1 practically always indicates thoracic disease. When abdominal pain is diffuse breathing is frequently costal though the abdomen usually moves with respiration. When the pain is localized the request to take a deep breath is likely to produce greater voluntary restraint of the abdominal movements and increased costal expansion.

In an endeavor to distinguish between thoracic and abdominal disease in children McClure studied the abdominal and thoracic respiratory movements by means of smoked drums. In almost 80 per cent of the cases of acute appendicitis he found the curve of abdominal movement lower than that of thoracic movement although in healthy children it is usually higher. The findings were reversed in 5 of 6 cases of pneumonia in which abdominal tenderness or rigidity or both were present. McClure concluded on the basis of these observations that restriction of the abdominal respiratory movement as indicated by pneumographic tracings is a valuable though by no means constant sign of acute appendicitis. The method is obviously not suitable for general use and if it is employed too much reliance must not be placed on the findings.

Examination of the chest early in thoracic disease usually reveals only diminished resonance. Later there may be crepitant rales and areas of dullness. The movement of the chest wall on the affected side is limited and pressure may cause pain. Roentgenologic examination will often reveal an early central pneumonia not otherwise demonstrable.

Thoracic disease may be responsible for abdominal pain particularly in young children if the parietal pleura is irritated. The pain which is seldom definitely localized is most pronounced along the distribution of the tenth, eleventh and twelfth intercostal nerves. It is likely to be absent unless the patient coughs or breathes deeply whereas

in abdominal disease pain is either continuous or spasmodic without reference to respiration. In thoracic disease the abdominal pain is usually associated with joint and muscle pains, brachialgic pain in the eyelids and other generalized pains not complained of in acute appendicitis. In thoracic disease pressure over the left iliac fossa with the fingers directed toward the right side does not produce pain in the right iliac fossa as it frequently does in acute appendicitis.

Rigidity of the abdominal wall is even more variable in thoracic disease than in acute appendicitis. It can often be overcome by deep palpation which does not increase the distress as it would if the pain arose from the peritoneal irritation associated with acute appendicitis. Palpation over an involved area in abdominal lesions increases rigidity since the deeper the palpation the greater the tenderness.

If after careful physical and roentgenologic examination it is still not possible to determine whether the disease is abdominal or thoracic it is safer to conclude that it may be abdominal and undertake exploration if only because of the possible co-existence of the two states. The diagnosis of abdominal or intestinal influenza so popular with the laity and with some physicians is a hybrid diagnosis which many times masks serious abdominal disease including rupture of the appendix with peritonitis.

*Cardiac Disease.* Various cardiac diseases may be associated with epigastric or even lower abdominal pain which may be quite severe as well as with nausea, vomiting and other symptoms and signs of acute appendicitis. In angina pectoris there is usually a history of previous attacks and the present attack is likely to have been precipitated by exertion. The patient complains of pain in one or both arms. He insists on sitting up. The face is pale and the eyes are wild. The appearance, posture and obvious fear of death are usually sufficient to distinguish the disease from acute appendicitis.

In acute cardiac disease with sudden decompensation the attack may begin with collapse. The liver is likely to be enlarged and tender as the result of passive congestion and palpation of the abdomen may give a sense of muscular resistance which must be distinguished from true rigidity. In acute pericarditis true rigidity is present but other signs of abdominal disease are lacking. In all instances of possible cardiac disease a competent internist should at once carry out a complete study of the cardio-respiratory system.

While confusion sometimes arises between acute cardiac disease and acute appendicitis an even greater danger lies in overlooking the development of appendicitis with mild symptoms in persons with

chronic long standing cardiac conditions In the following case for instance all the attention was concentrated on the cardiac state and an abscess which pointed externally was actually drained without any realization of its appendiceal origin

*Case 7* A 42 year old colored woman who had been in the hospital for 5 months with syphilitic heart disease complained of pain in the right lower abdomen but had no other symptoms Ten days later a mass presented in the right inguinal region it was incised a week later after it had become fluctuant During this period the temperature was 100 F and bronchopneumonia was suspected but the diagnosis was not confirmed Death occurred 2 months after the initial abdominal symptoms from acute cardiac decompensation Postmortem examination disclosed in addition to the anticipated cardiac disease a rupture of the appendix and generalized purulent peritonitis the appendix had completely sloughed off and lay free in the peritoneal cavity

*Gastro intestinal Disease* In addition to bearing in mind the fact that acute appendicitis may occur coincident with dietary indiscretions it is necessary also to distinguish between acute appendicitis and gastro enteritis Any type of food poisoning may cause abdominal pain vomiting diarrhea fever and sometimes collapse Often the patient seems more acutely ill than a patient with acute appendicitis Rigidity of the abdominal wall and tenderness are however usually lacking In so called ptomaine poisoning there is a history of eating tainted food and several persons may be attacked simultaneously Individual dietary indiscretions are more difficult to evaluate and the fact that they have occurred as has been pointed out does not in any way exclude the diagnosis of acute appendicitis Even more confusion is precipitated when an apparent dietary disturbance follows the ingestion of food which would not ordinarily cause difficulty as is shown by the following case The final diagnosis was gastro enteritis but the history and findings were typical of acute appendicitis in its classical form and operation was done chiefly because the appendix could not be excluded as the cause of the symptoms

*Case 8* A 36 year old white man developed cramping pain to the left of the umbilicus 3 hours after lunch Later the pain shifted to the right of the umbilicus and still later to the right lower quadrant Nausea occurred soon after the onset of pain and the patient vomited 4 times He also had several loose bowel movements Both breakfast and lunch had been eaten at home and other members of the family who remained well had partaken of the same simple food (milk and sandwiches of ham baked at home) Six hours after the onset of abdominal pain the temperature was 100 F the pulse 84 and the respiration 20 per minute There was increased resistance over the

entire right side of the abdomen and point tenderness and muscle spasm were present over McBurney's point. Rectal examination disclosed right sided tenderness.

Operation through a right rectus incision under ethylene anesthesia revealed a grossly normal retrocecal appendix; there was no evidence of Meckel's diverticulum or of any other abnormality. The appendix was reported as entirely negative. Recovery was uneventful.

The diagnostic difficulties in the following case which is another instance of atypical acute appendicitis in middle life arose from the apparent dietary origin of symptoms in a heavy drinker and the absence of localized tenderness throughout the illness.

**Case 9.** A 54 year old white man known to be a heavy drinker developed pain in the right lower quadrant of the abdomen following a large Thanksgiving dinner. Later the pain became generalized and nausea and vomiting occurred. When the patient entered the hospital 24 hours after the onset of symptoms he was greatly dehydrated. The temperature was 99.2° F, the pulse rate 84, the respiratory rate 20, and the blood pressure 160/90. The abdomen was slightly distended and there was generalized tenderness and moderate generalized rigidity. The white blood cell count was 12,500 with 82 per cent polymorphonuclear leukocytes.

After 4 days of symptomatic treatment on the medical service the patient was transferred to the surgical service with a diagnosis of ruptured peptic ulcer. Roentgenologic examination of the abdomen at this time showed multiple fluid levels but no free gas. The nonprotein nitrogen of the blood was 87 mg per cent. The recurrent colicky pain suggested to the surgical resident the diagnosis of volvulus and the abdomen was therefore opened at once under spinal analgesia through a low midline incision. A purulent peritonitis was drained without any effort to determine the source of the infection. The patient died 3 hours later. Autopsy revealed a rupture of the appendix and generalized purulent peritonitis.

Less severe types of digestive disturbances than those illustrated are sometimes related to the mere ingestion of food, sometimes of special articles which have previously disagreed with the patient. Tenderness, localized pain, and fever are not likely to be present in such cases. Repeated attacks diagnosed as bilious attacks (especially in children), indigestion, gastritis, and food idiosyncrasies sometimes prove to be acute appendicitis. The particular risk is that if the symptoms are accepted as of digestive origin when the patient is not severely ill, they are likely to be accepted as such in more serious attacks. As a result the examination is incomplete and the diagnosis of acute appendicitis may be missed with disastrous consequences.

**Intestinal Infections** A number of writers of whom Aschoff was among the first have pointed out that parasitism may cause pain and other symptoms suggestive of acute appendicitis Swartzwelder for instance found numerous such cases in a study of clinical *Trichocephalus trichiurus* infection at the New Orleans Charity Hospital and Hinman found similar cases in a study of strongyloidiasis at the same institution

Acute appendicitis is much less often confused with amebiasis than is chronic appendicitis which is fortunate for the mortality associated with appendectomy under the circumstances is very high The widespread incidence of amebiasis in the general population is now established and the appendectomies performed on incorrect diagnoses during the Chicago epidemic of amebic infection are indicative of the confusion that may arise

In 52 personally observed cases of amebiasis reported by Massie abdominal pain was a symptom in 49 Frequently the pain radiated down the leg Frequently it was localized in the right lower quadrant of the abdomen and was associated with local tenderness A number of patients had been operated on previously for supposed appendicitis but fortunately in no case was the amebic infection of the acute fulminating variety

Differential diagnosis cannot be made clinically but depends on the identification of cysts of *Endamoeba histolytica* in the stool Appendectomy is occasionally necessary when acute appendicitis and amebiasis are actually coincident but whenever possible according to Ochsner and DeBakey conservative therapy should be employed Any operative manipulation is undesirable and dangerous because the amebic infection practically always involves the cecum as well as the appendix If appendectomy is performed without realization of the amebic disease the patient's chances of recovery as Gilje and Lampson have pointed out depend upon intensive anti amebic therapy and upon complete rest of the intestine to permit healing of the appendiceal stump

Rubenstein and Johnson called attention to the syndrome suggestive of acute appendicitis sometimes associated with Salmonella infections They observed this syndrome in 20 of 811 patients with this type of infection though in only 7 of the 18 patients who came to operation was the appendix acutely diseased or gangrenous This is in contrast to other reported series in practically all of which the appendix was either acutely inflamed or had ruptured In a postoperative evaluation of preoperative symptoms and signs Rubenstein and Johnson could find no clue in the clinical picture which might have prevented unnecessary surgical intervention The best they could suggest is that the persistence

of unexplained fever after appendectomy should raise the question of a possible *Salmonella* infection. The organism is not likely to be identified in the excised appendix unless special cultural studies are made.

**Malaria** The possibility of confusing appendicitis and malaria is particularly great in a region in which malaria is endemic. The wartime experience of Ravdin and North in the China Burma India Theater of Operations substantiates this statement. They observed 10 cases in which the two diseases occurred simultaneously and in 7 of which the appendicitis was acute diffuse suppurative or worse. In a review of 10818 appendectomies at the New Orleans Charity Hospital Copland and Miangolarra found 12 cases in which operation had been performed in error the real disease being malaria. In a review of 842 cases of malaria they found 31 in which the symptoms were typical of acute appendicitis. They therefore concluded that it is malaria rather than appendicitis which does the masquerading.

The differential diagnosis rests on the demonstration of plasmodia in the blood stream. Clinical differentiation is not reliable. The initial temperature elevation is usually higher in malaria than in appendicitis and the white blood cell count is likely to be lower. Pain is a constant symptom in the pseudo appendiceal syndrome which occurs in malaria but tenderness and rigidity though fairly frequent are inconstant. Chills regularly occur in malaria but they are by no means infrequent in acute appendicitis (p. 119). The history of previous attacks of either malaria or appendicitis may be helpful but is not conclusive.

If the condition is acute exploration must be undertaken on the basis that acute appendicitis cannot be positively excluded. Ravdin and North's point is well taken that whenever a large group of individuals with malaria is concentrated as it was in the China Burma India Theater during World War II it is quite possible that some of them will also present acute abdominal disease.

**Typhoid Fever** Typhoid fever is not infrequently accompanied by abdominal pain which may be localized in the right iliac fossa. Considerable diagnostic confusion may be caused by this syndrome in a patient with the ambulatory type of disease who is unaware that he has had fever. Rouhier described a peculiarly serious form of typhoid fever observed in French soldiers in World War I in which the first manifestation of illness was due to perforation of the appendix. In the more typical case of typhoid fever the irregular fever headache general malaise slow pulse with dicrotic wave splenic enlargement and roseola usually make the diagnosis clear even before it is established.



by laboratory methods. If perforation of the ileum occurs the differential diagnosis is practically impossible.

It is quite possible for acute appendicitis to occur simultaneously with typhoid fever and for an extremely confusing situation to arise. In a case of this sort observed at Charity Hospital the interne on the medical service (to whom appendicitis and its dangers had been particularly emphasized in his student days) insisted that a patient with proved typhoid fever also had acute appendicitis. After several hours he brought the staff to his point of view and surgical consultation was requested. At operation the appendix was found to be perforated. In the following case the outcome was less happy. The initial symptoms of acute appendicitis and of typhoid fever probably coincided. The diagnosis of subsiding acute appendicitis was incorrect as it frequently is in patients in middle and late life; the residual cul de sac abscess was not recognized and the diagnosis of subphrenic infection was also incorrect as it frequently is.

*Case 10.* A 42 year old white man complained for 72 hours of bilateral subcostal pain, more marked on the right side, accompanied by chills. The temperature was 104° F, the pulse rate 120 and the respiratory rate 30. Rigidity and muscle spasm were not present and a diagnosis of subsiding acute appendicitis was made on the basis of suggestive rebound tenderness. Tests for malaria were negative. The white blood cell count was 12,000 per cu. mm. with 90 per cent polymorphonuclear leukocytes.

Forty-eight hours after the patient was hospitalized a mass could be palpated in the right lower quadrant of the abdomen and the standard routine for peri-appendiceal abscess was instituted, including the oral and parenteral administration of a sulfonamide drug. On the tenth day of hospitalization the abdomen was opened under ether anesthesia through a McBurney incision. The appendix was gangrenous and there was a localized abscess. Appendectomy and drainage were carried out and powdered sulfanilamide was placed in the peritoneal cavity.

The pre-operative routine was continued after operation. Chemotherapy was discontinued on the tenth day when blood was found in the urine. On this day stool examination and agglutination tests were positive for typhoid fever. On the twenty-fifth day the subphrenic space was explored with negative findings and on the twenty-ninth day an expected residual abscess failed to materialize when the abdomen was reopened. The patient died the following day. Autopsy revealed a cul de sac abscess, pyelonephritis, acute suppurative cholecystitis, bronchopneumonia, fatty metamorphosis of the liver and hepatic edema.

*Tubes Dorsalis.* It would seem impossible to confuse the gastric crists of tubes dorsalis with acute appendicitis except that this has been

done in a number of recorded cases. The classic report by Nuzum concerns 97 operations performed on 87 (of 1000) patients with tuberculous dorsalis studied at the Cook County Hospital the pre operative diagnosis in 18 cases being acute appendicitis.

If complete examination is carried out the diagnosis of tuberculous dorsalis can usually be established without difficulty by the absence of fever tenderness and rigidity and elevation of the white blood cell count by the presence of characteristic pupillary changes (Argyll Robertson pupil) by the impairment or complete absence of the patellar reflexes and by a past history of syphilitic disease or antisyphilitic therapy. Acute appendicitis of course should not be excluded too hastily it is entirely possible for a tuberculous subject to have that disease also.

*Occupational Poisoning* The differential diagnosis of lead colic is made by the absence of fever and leukocytosis the scaphoid abdomen the peculiarly obstinate constipation the characteristic blue line on the gums and stippling changes in the red blood cells. Inquiry into the occupation would seem to settle the matter workers on lead plate storage batteries as well as painters are susceptible to the disease. It is of course possible to overlook acute appendicitis in a painter if too much emphasis is placed on the occupation.

*Case 11.* A 17 year old white painter had been treated at home for 17 days for periumbilical pain moderate abdominal distention nausea and vomiting chills and drowsiness. The temperature when he was hospitalized was 97° F the pulse rate 80 and the respiratory rate 20. Physical examination revealed generalized abdominal tenderness and rigidity. The white blood cell count was 2500 per cu mm with 67 per cent polymorphonuclear leukocytes. Roentgenologic examination revealed air under both leaves of the diaphragm. At operation which was performed at once the appendix was found ruptured and gangrenous and a quart of purulent fluid was removed from the peritoneal cavity. The patient died in wild delirium 24 hours later.

*Peritonitis of Nonappendiceal Origin* Tuberculous peritonitis is usually of more gradual onset than peritonitis following acute appendicitis acute appendicitis in the early stages is unlikely to be confused with it. Confusion is also unlikely in the ascitic type in which a fluid wave is clearly demonstrable. In the inflammatory or dry type it is quite possible to mistake one of the intestinal masses for an appendiceal abscess particularly if ulceration has caused acute symptoms though in such a case other masses can usually be felt. If the previous history is inquired into it will be found that the patient even if pul

monary tuberculosis has not been diagnosed is likely to have lost weight and strength and to have suffered from fever night sweats cough malaise and anemia. A low white blood cell count is the rule in tuberculous peritonitis. Operation does no harm if the diagnosis of acute appendicitis proves incorrect for the mere opening of the abdomen for some unexplained reason often results in at least temporary improvement in tuberculous peritonitis.

It must be remembered that a patient with pulmonary tuberculosis can also develop acute appendicitis. One of the patients in the non-surgical group of cases at Charity Hospital of Louisiana at New Orleans who had been in the hospital for many months with pulmonary tuberculosis developed acute appendicitis and died of it without ante-mortem recognition of her condition.

Pneumococcic peritonitis is most frequent in children and young women. It is a rather common sequel of chronic glomerular nephritis. It frequently begins with diarrhea or vomiting high fever and marked prostration. Tenderness is generalized and there is no history of epigastric or umbilical pain with later localization as there is likely to be in acute appendicitis. There is resistance rather than rigidity of the abdominal wall usually associated with moderate distention. The white blood cell count is always high and may be over 30 000 per cu mm. Pneumococcic peritonitis is not benefited by operation and every effort therefore should be made to establish the definite diagnosis.

Streptococcic peritonitis is frequently preceded by symptoms of acute sore throat which are just beginning to subside when the patient again becomes acutely ill with abdominal pain anorexia vomiting fever rapid pulse abdominal tenderness and moderate rigidity. The differentiation is always difficult and may be impossible in the absence of exploratory laparotomy.

*Exanthematous Diseases*—French physicians have attached importance to a supposed relationship between acute appendicitis and scarlet fever but Ronaldson after a study of approximately 27 000 patients with scarlet fever concluded that the joint occurrence of the two diseases is merely coincidental. At the Willard Parker Hospital Bullowa and his associates observed only 3 cases of acute appendicitis over a 5 year period among 252 cases of scarlet fever.

The same observers over the same period observed 11 cases of acute appendicitis among 6357 cases of measles in children between the ages of 3 and 13 years in every instance perforation had already occurred and 2 cases terminated fatally. Two patients developed acute appendicitis during the prodromal stage of measles and 9 during the

post-eruptive stage Hudson and Krikower who observed 8 cases of acute appendicitis during the course of measles over a 5 year period at the Children's Hospital in Boston pointed out that up to 1931 only 31 cases had been recorded in the literature since the first case was reported in 1901 Five of their patients who were operated on at intervals between 7 and 13 days after the onset of illness had well localized abscesses

Bullowa and his associates called attention to a specific giant cell reaction characteristic of the prodromal stage of measles which was observed in 3 of the 4 appendices in their series removed during the pre-eruptive stage of measles In their opinion this reaction which was present in the mucosal and submucosal layers of the appendix indicates that the appendix is involved directly or indirectly in the exanthematous disease Hudson and Krikower who compared sections from the appendices removed in their 8 cases of measles with sections from the appendices of 200 children without measles found in the measles cases generally less lymphoid tissue and practically no secondary centers or germinal follicles In the control group the germinal follicles were generally large and prominent The appendices from the cases of measles generally presented more plasma cells; especially in the submucosa In 2 cases numbers of larger cells were observed in the mucosa lymphoid tissue and submucosa with basophilic cytoplasm and large prominent nuclei The nuclei were often oval and spherical and were sometimes lobated and distorted In occasional instances there were two and even three nuclei Cells of this kind were infrequent in the mucosa in the control cases and extremely infrequent in the submucosa and lymphoid tissue

The relation if any which exists between measles and acute appendicitis remains to be clarified The important consideration at this time is that acute appendicitis which develops coincident with measles and which frequently is of a fulminating character shall not be overlooked Since a pseudo-appendiceal syndrome is rather frequent in the prodromal stages of measles and other exanthemata this is not always a simple task Bullowa and his associates reported that over the period of their study 4 patients were transferred to the Willard Parker Hospital from other municipal institutions because measles had appeared a few days after appendectomy had been performed All 4 patients had presented the classical picture of acute appendicitis but in no instance was the appendix abnormal enough either grossly or histologically to have warranted operation

*Mesenteric Lymphadenitis* From the interest displayed in the subject

in the recent literature one might suppose that the incidence of mesenteric lymphadenitis is increasing it seems more reasonable to assume that the disease is merely being recognized more frequently In the 147 cases reported by Foster from the New York Post Graduate Hospital over a period from 1914 to September 1937 24 had occurred in the last 13 months of the study Aird observed 37 cases in a single year at the Royal Edinburgh Hospital for Sick Children and over the same period observed 83 instances of acute appendicitis

The disease is most frequent in children and young adults its greatest incidence thus coinciding with the greatest incidence of acute appendicitis It most often occurs in poorly nourished and tuberculous subjects

The symptoms and findings are variable Usually there is a history of an acute upper respiratory infection within a week or so of the acute attack or a history of preceding attacks suggestive of atypical acute appendicitis The pain is uniformly distributed through the lower abdomen and is steady continuous and dull Nausea is frequent but vomiting is not Constipation is the rule Occasionally the onset is stormy with high fever prostration vomiting a greatly elevated white blood cell count and abdominal distention The outlook is poor in such cases in contrast to the excellent prognosis in the usual case

Physical findings include tenderness chiefly about the umbilicus and extending into the right lower quadrant or less often to the left Occasionally tenderness is bilateral All lymph nodes including the abdominal nodes are enlarged Sometimes the descending colon can be palpated as a doughy coil Distention is frequent but rigidity and spasm are unusual If rebound tenderness rigidity and spasm are present an associated acute appendicitis must be assumed the association is not unusual The most useful diagnostic maneuver is to endeavor to elicit tenderness along the root of the mesentery that is in a line extending from the right lower to the left upper quadrant with the umbilicus as the central point

Aird stated that the differentiation between acute nonspecific mesenteric lymphadenitis and acute appendicitis is so simple that he confidently permitted children with the former disease to leave the hospital as soon as the diagnosis was made others are less positive His points of differentiation are as follows Tenderness in the right lower quadrant is higher and more medial than in acute appendicitis The lowest limit is to the right of and just below the umbilicus When the superior mesenteric glands are widely involved a band of tenderness can be outlined extending upward and to the left toward and

sometimes just beyond the midline of the epigastrium. The abdominal muscles in the lower right quadrant are nearly always in slightly higher tone than on the left but the rigidity is either not marked or will disappear under continued gentle pressure. Rebound tenderness is not unusual particularly if the leaves of the mesentery or the serosa of the bowel are inflamed. Aird has never himself elicited pain in the right lower quadrant by palpation on the left side. He seeks the site of maximum tenderness while the patient lies supine then changes the position to the left lateral. In this position after a few minutes the point of maximum tenderness will be found to have shifted to the left sometimes beyond the midline. The sign depends upon postural displacement of the lower ileum with its mesentery and can be elicited only if the regional glands are inflamed. It is occasionally positive early in acute appendicitis if the cecum is highly mobile.

The appendix seems associated in some way with acute mesenteric lymphadenitis even if acute appendicitis is not present it is generally agreed that the percentage of patients relieved of all symptoms after appendectomy is too high to be explained by mere coincidence. Aird however dissented from this point of view. Foster's suggestion that at operation the intestines be removed from the peritoneal cavity and exposed until a considerable hyperemia develops obviously should not be employed if acute appendicitis is also present.

*Miscellaneous Nonsurgical Conditions* While it would be impossible to discuss all of the nonsurgical conditions which may mimic acute appendicitis some of the more unusual might be briefly mentioned. Wechsler pointed out the possibility of acute or subacute symptoms referable to the abdomen in association with certain cerebral lesions. Moore described paroxysmal abdominal pain as a form of focal symptomatic epilepsy and reported a case in which appendectomy was performed in the course of an attack. Butsch and Harberson over a 6 month period in service observed 50 patients with acute virus infection with nerve involvement simulating appendicitis 18 of whom were submitted to appendectomy before the cause of the pain was understood.

Beardwood in a study of 1000 consecutive patients with diabetes found 114 in whom abdominal symptoms were pronounced and 14 who required surgery for acute abdominal disease 5 instances of appendicitis were included in the latter group. Neiman who found 8 cases of acute appendicitis only 1 of which was unruptured among 1251 cases of diabetes pointed out that diabetic acidosis may be accompanied by diffuse abdominal pain and tenderness the onset of which

■ insidious In his opinion if symptoms persist after adequate insulin therapy over a period of not more than 6 hours operation should be done in any case in which acute appendicitis cannot be excluded Langmann called attention to the pseudo appendiceal syndrome which may occur at the onset of acute rheumatic fever in young children and pointed out the importance of differentiating this syndrome from true acute appendicitis which may develop in the course of the illness

Bowers and Richard as well as Babbage and his associates called attention to the possible mimicry of acute appendicitis by a strain of the right rectus muscle The former authors observed ■ such cases and the latter 141 such cases all in men in service over relatively short periods of time Cullen and Brodel had previously written extensively on this same diagnostic difficulty

In both acute appendicitis and strain of the right rectus muscle the picture is likely to include pain in the lower abdomen nausea vomiting a moderate elevation of temperature a moderate leukocytosis and rebound tenderness Spasm of the rectus muscle is not always present in muscle strain but it is not uniformly present in acute appendicitis either Diagnosis in the cases mentioned was greatly simplified by the fact that under military conditions patients could be hospitalized without delay and observed at frequent intervals The presence of ecchymosis or of a hematoma in the muscle though not constant readily permitted exclusion of acute appendicitis when either was present Babbage and his associates also found that the injection of procaine hydrochloride into the rectus muscle was useful diagnostically as well as therapeutically it almost immediately relieved the distress of muscle strain but had no influence on pain and tenderness due to acute appendicitis

### URINARY TRACT DISEASE \*

In an abdominal condition with urinary symptoms which may be acute appendicitis the first diagnostic consideration is whether the symptoms are part of the syndrome of appendicitis or are really due to urinary tract disease As has been pointed out (p 116) acute appendicitis may be associated with such urinary symptoms and signs as

\* The importance (and difficulty) of the differential diagnosis of acute appendicitis and urologic diseases is shown by the 168 page book recently published on the subject (Strominger L. *Appendicite et Urologie Etude Medico Chirurgicale Clinique et Therapeutique* Masson et Cie Paris 1946) I have not had an opportunity to examine this book but it is commended highly by J E Semple in his review in the *British Medical Journal* [2 337 (30 August) 1947]

frequency dysuria retention pain in or retraction of one or both testicles and hematuria. It is not always possible to determine whether abdominal pain nausea and vomiting are the major symptoms with the urinary symptoms subsidiary or whether the reverse is true and the urinary disease is primary.

Diseases of the urinary tract which may simulate acute appendicitis include chiefly pyelitis; perinephric abscess renal and ureteral colic hydronephrosis diverticulum of the bladder and cystitis.

*Pyelitis* The onset of pyelitis which is most frequent in women and children is frequently stormy with chills and high fever. The initial pain although it may be extremely severe is usually less sudden and sharp than in acute appendicitis. It is most often located in the lumbar region. Tenderness is constant but rigidity is seldom present. The leukocytosis is likely to be higher than in appendicitis. Hematuria may or may not be observed but pus and bacteria are almost always found in the urine. Diagnosis is established by the history and by examination of the urine. If the kidney can be grasped between the hands local pain is caused and the Murphy maneuver of striking the kidney region with the fist will always elicit an active response from the patient with pyelitis.

*Perinephric Abscess* Perinephric abscess is usually insidious in origin as compared to the abrupt origin of most cases of acute appendicitis. Roentgenologic examination in the lateral position may show the kidney pushed forward by the abscess. Retrograde pyelography with a ureteral catheter in place on the right side is also useful. Anteroposterior and lateral views should be taken.

Perinephric abscess is extremely difficult to distinguish from rupture of a retrocecal appendix with abscess formation particularly if the initial symptoms of acute appendicitis have been atypical. In the following case the mistaken diagnosis was due to the atypical location of the pain as well as to the urinary symptoms and signs. The unusual spread of the infection could be attributed to the retrocecal position of the appendix.

*Case 12* A 7 year old white girl had been ill for 8 days when she was first seen. The illness was initiated by pain in the right lumbar region below the costal margin which later shifted to the right of the umbilicus and then gradually subsided. Frequency of urination and nocturia had been present throughout the illness. The temperature was 102° F. the pulse rate 120 and the respiratory rate 24. Physical examination revealed rigidity and tenderness in the right lower quadrant of the abdomen and also in the right lumbar region where a possible mass was felt. Rectal examination was negative. The



urine contained pus (4 plus). The white blood cell count was 33 000 per cu mm with 69 per cent polymorphonuclear leukocytes.

Operation performed on the diagnosis of perinephric abscess revealed at the lower pole of the right kidney a mass of densely adherent infected tissue which proved to be an abscess cavity containing a fecalith. Drainage was instituted. The patient died 18 hours later. Autopsy revealed a ruptured appendix fibropurulent peritonitis and a retrocecal phlegmon.

**Renal and Ureteral Colic.** Renal and ureteral colic in their typical forms are much more severe than acute appendicitis even of the obstructive variety. They are frequently, however, atypical. In the typical case the pain and tenderness are definitely located in the renal area. In the atypical case they may be located anywhere in the abdomen including the right iliac fossa. The pain of renal (or ureteral) colic is likely to become maximal shortly after the onset of an attack and to continue at a maximal level whereas in acute appendicitis the initial pain is likely to be spasmodic and is seldom as excruciating. Hematuria is present in most—though not all—cases of renal or ureteral stone but it may also be present in acute appendicitis. Later evidences of inflammatory changes such as fever and leukocytosis eventually appear in acute appendicitis but are absent in renal and ureteral colic in which the pain is often out of proportion to the other symptoms though rigidity is never present.

Differentiation is most difficult in the cases of acute appendicitis in which right scrotal pain is present. In suspected cases of urinary tract calculi roentgenologic examination with a right ureteral catheter *in situ* usually establishes or rules out the presence of a stone. Cystoscopic examination is also helpful. Levitas described a series of tender points diagnostic of ureteral disease and serving to distinguish it from retrocecal appendicitis in which the points of tenderness are in other locations.

**Hydronephrosis.** Hydronephrosis may cause pain resembling renal colic. In the intermittent type of case the pain usually reaches its maximum level promptly. Sometimes it is dull and persistent. Often a rounded tense tender swelling occupies the lateral aspect of the abdomen and can be felt back into the groin. It is larger than the usual appendiceal abscess and is less tender. There may be a history of oliguria or a history of previous similar attacks in which symptoms were relieved when the patient suddenly passed a large quantity of urine with prompt disappearance of the swelling. A pyelogram usually establishes the diagnosis. If pyonephrosis develops there are constitutional

disturbances such as fever leukocytosis and toxemia and pus may appear in the urine.

*Cystitis* Cystitis is differentiated from acute appendicitis by urinalysis and by the fact that the patient complains of pain not at the beginning of urination but at the end when the bladder is empty and its inflamed surfaces meet. In acute appendicitis the symptoms are likely to be continuous or the pain occurs at the beginning of urination so that the patient tries not to void. The symptoms of diverticulum of the bladder are substantially the same.

*Infection of the Perirenal Fat* Goldstone and LeMarquand in the course of World War II observed a new syndrome due to acute staphylococcal infection of the perirenal fat which in some instances mimicked acute appendicitis.\* Differentiating points were the usual prominence of signs in the right upper abdominal quadrant and the usual absence of the sequence of umbilical pain, nausea or vomiting and localized pain in the right iliac fossa.

### GYNECOLOGIC STATIS

Various gynecologic conditions must be differentiated from acute appendicitis including dysmenorrhea, threatened abortion, intermenstrual pain, rupture of a Graafian follicle or lutein cyst, salpingitis, a twisted ovarian cyst or tumor, a twisted ovarian fibroid and ectopic pregnancy. Some of these conditions themselves demand immediate surgery and in all it is quite possible for acute appendicitis to coexist with the gynecologic condition.

*Dysmenorrhea* Dysmenorrhea is distinguished from acute appendicitis by the periodicity of the pain, its usual reference to the lower lumbar, sacral and pelvic regions and the absence of a constitutional reaction. The frequency of acute appendicitis combined with the periodicity of menstruation makes it inevitable that the onset of the two conditions should frequently coincide (far more often as a matter of fact than the statistics from the New Orleans Charity Hospital suggest) and should occasionally give rise to diagnostic difficulties. The mere fact that a patient has never before had dysmenorrhea does not necessarily exclude that diagnosis though it does support a tentative diagnosis of acute appendicitis.

*Ovarian Conditions* The most usual ovarian conditions which simulate acute appendicitis are ruptured Graafian follicles, corpus

\* Simulation of acute condition in abdomen by infection of perirenal fat (Foreign Letters) J. A. M. A. 132:469 (26 October) 1946.

luteum cysts and bleeding corpora lutea. When operation is done in any of these conditions it is usually on the diagnosis of acute appendicitis. It is also possible as McLaughlin's and Bernstein's reports indicate for acute appendicitis and rupture of a Graafian follicle to occur simultaneously.

Differentiation is made on the following points: (1) In the ovarian lesion the pain is always local whereas in acute appendicitis it is first generalized and then localized. In the ovarian lesion nausea is less frequent than in acute appendicitis and vomiting is unusual. In the ovarian lesion spotting occasionally occurs whereas there is no spotting or discharge in acute appendicitis. In the ovarian lesion the tenderness in the lower abdomen is slight and there is no rigidity whereas in acute appendicitis there is definite tenderness at McBurney's point and rigidity is frequently present. The temperature and pulse rate are normal in the ovarian lesion but both are likely to be elevated in acute appendicitis. In the ovarian lesion the white blood cell count and polymorphonuclear leukocytes are at the upper limit of normal in acute appendicitis both are moderately elevated. Pelvic tenderness or a doughy sensation elicited on pelvic examination suggest that the condition is a ruptured follicular or lutein cyst. Obviously the diagnosis would not be so often missed if rectal or vaginal examination were carried out routinely in women with supposed acute appendicitis.

Bernstein's suggestion that a 24 hour period of careful observation may prevent an unnecessary operation must be weighed against the disaster which might follow such delay if the lesion proved to be acute appendicitis.

**Intermenstrual Pain.** Intermenstrual pain (*mittelschmerz*) probably occurs in more cases than is realized because according to Wharton and Henriksen patients do not contribute information about mild types of discomfort and physicians fail to ask specific questions concerning it. The mild type of pain however is unlikely to be confused with acute appendicitis partly because of the mildness of the discomfort but chiefly because of its striking periodicity. In acute cases on the other hand the differentiation is much less clear cut. Half of the 61 cases reported by Wharton and Henriksen from clinic and private practice were of the acute type so severe that they could not be missed and 21 emergency operations had been done in this group chiefly on the diagnosis of acute appendicitis. In the acute cases the periodicity of the pain was by no means as striking as in the mild type.

Wharton and Henriksen have outlined the following differentiating points. In *mittelschmerz* there is a history of previous similar attacks at

regular and sometimes at periodic intervals with prompt convalescence. The pain in the dysmenorrhœic variety in spite of its severity. Leukorrhœa and vaginal bleeding are sometimes present and vaginal washings will often reveal microscopic blood. Symptoms referable to the bladder may be present. Gynecologic examination shows tenderness of the entire pelvis usually more marked on one side than the other the findings are sometimes suggestive of acute salpingitis. There may be a sense of induration on the affected side though tenderness usually prevents outlining of the ovaries. Many of these symptoms and signs it will be noted are present in acute appendicitis also and when the clinical picture is suggestive of that disease especially when nausea vomiting fever and leukocytosis are present it would require extreme self confidence as Wharton and Henriksen concluded to advise any thing but exploration.

*Pelvic Inflammatory Disease* Pelvic inflammatory disease must frequently be differentiated from acute appendicitis in communities with a large Negro population such as New Orleans. Diagnosis is greatly simplified if a reliable history can be secured of previous acute attacks of abdominal distress associated with urinary disturbances menstrual disorders a vaginal discharge and other evidence of venereal infection even if a definite history of exposure is lacking.

In a typical case of pelvic inflammatory disease the pain is usually gradual in onset bilateral confined to the pelvis and persistent. Back ache is frequent. Tenderness at McBurney's point is not elicited and muscular rigidity is either absent or not marked. Distention if it is present is confined to the lower abdomen. Hyperesthesia is either absent or is located at a point considerably below its location in appendicitis. The temperature pulse rate and leukocytosis are generally higher in pelvic inflammatory disease than in appendicitis though after the first few hours the patient looks less ill than the patient with appendicitis. The sedimentation rate is not a reliable means of differentiation (p 134).

The onset and course of an attack of salpingitis may be entirely atypical. If suppuration is present the tenderness may extend quite high in the abdomen. The attack may be associated with or may follow closely upon a menstrual period but for that matter so may an attack of appendicitis. The presence of a vaginal discharge though significant does not necessarily exclude the diagnosis of acute appendicitis. Pelvic examination is usually conclusive. The uterus is likely to be fixed by tender bilateral masses and there is tenderness on pressure in the cul de sac especially on digital movement of the uterus in the absence of pressure by the abdominal hand.

A ruptured pyosalpinx is not usual but if it does occur it must be distinguished from ruptured appendicitis with peritonitis. In late cases this may be possible only by the history. In early cases there may be confusion between a pelvic abscess due to tubal disease and rupture of a pelvic appendix. Salpingitis frequently causes periappendiceal disease which does not usually give rise to symptoms suggestive of acute appendicitis.

Sattler (cited by Royster) suggested as a diagnostic maneuver that the patient be asked to sit up in bed with the right leg raised and extended if necessary with the aid of the examiner. The cecum is thus pressed between the abdominal wall and the psoas muscle. In acute appendicitis the maneuver causes sharp pain at the junction of the right and middle third of a line joining the iliac spines. In salpingitis there is either no pain or it is further to the right.

These differential points are all important and useful but they are not always conclusive. An analysis of 60 so called unnecessary emergency operations at the New Orleans Charity Hospital over a recent 3 year period proves that point. The pre-operative diagnosis in every instance was acute appendicitis but the finding in every instance was pelvic inflammatory disease. A review of the clinical picture suggests that the pre-operative diagnosis was justified in almost every case. It is significant that not a single patient in the series complained of back ache which is a common symptom in pelvic disease and which it might be well to bear in mind as an important diagnostic point.

Operation is not desirable in acute pelvic disease but exploration may be necessary if acute appendicitis cannot be excluded. If the abdomen is opened in error, it should be closed without surgery. That the error causes no serious consequences is shown by the fact that in the 60 unnecessary explorations just referred to there were no fatalities and recovery was uncomplicated in almost every instance.

How difficult the differential diagnosis of pelvic inflammatory disease may be is shown by the following cases.

*Case 13.* A 21 year old Negro woman had suffered from lower abdominal pain, nausea and vomiting for 72 hours. She had had several similar attacks and had had a vaginal discharge for a year. The temperature was 99.6° F, the pulse rate 120 and the respiratory rate 30. Physical examination revealed generalized abdominal tenderness, most marked below the umbilicus and in both lower quadrants. Pelvic examination revealed extreme tenderness in both adnexal regions. Rectal examination revealed bilateral tenderness. Routine treatment for acute pelvic inflammatory disease was instituted. The patient died within 24 hours with an *antemortem* temperature (rectal) of 107° F.

Autopsy revealed rupture of the appendix generalized peritonitis fatty metamorphosis of the liver bronchopneumonia and pulmonary edema

The patient's history of leukorrhea the bilateral adnexal tenderness and the lack of localizing signs led to a justifiable but mistaken diagnosis of pelvic inflammatory disease. Death occurred so soon after admission to the hospital that it is doubtful whether any treatment would have altered the outcome.

*Case 14* A 13 year old Negro girl had been ill for 5 days with bilateral lower abdominal pain and frequent painful urination. A vaginal discharge had been present since the onset of illness. She had vomited once. The temperature was 100.8 F the pulse rate 132 and the respiratory rate 32. Rectal examination was negative but vaginal examination revealed bilateral adnexal tenderness. Physical examination revealed bilateral tenderness and rigidity. The white blood cell count was 9700 with 80 per cent polymorphonuclear leukocytes. The sedimentation time was 2 hours. Vaginal and urethral smears were negative.

The admission diagnosis of acute pelvic inflammatory disease was changed within a few hours to rupture of the appendix with generalized peritonitis which laparotomy under spinal analgesia through a McBurney incision proved to be correct. Appendectomy cecostomy and drainage were carried out. The patient was extremely toxic when she was first seen and death occurred 24 hours after operation from peritonitis and toxemia.

It is doubtful in the face of this girl's overwhelming toxemia that anything could have saved her life in an era when replacement therapy was not employed routinely or rationally and when chemotherapy antibiotics and intestinal decompression had not yet been introduced.

*Case 15* A 22 year old Negro woman had been ill for 3 weeks with leukorrhea backache lower abdominal pain and malaise. Three days before she entered the hospital she became acutely ill with nausea vomiting and fever superimposed on her previous symptoms. The temperature was 101 F the pulse rate 90 and the respiratory rate 24. Physical examination revealed generalized abdominal tenderness more marked on the lower right side and moderate abdominal distention. Pelvic examination revealed bilateral adnexal tenderness and a typical frozen pelvis. The white blood cell count was 12500 per cu mm.

Treatment was by the usual routine for acute pelvic inflammatory disease including transfusion. On the fifteenth day of hospitalization a mass was felt in the cul de sac. Four weeks later the patient developed bilateral parotitis which was treated by roentgen rays. A week later she developed an abscess of the right buttock. Numerous consultants could throw no light on the con-

dition. Positive blood cultures were never secured but a tentative diagnosis of gonococcal septicemia was made. Death from toxemia and exhaustion occurred on the sixty seventh day of hospitalization. Autopsy revealed rupture of a retrocecal appendix and dissecting retroperitoneal appendiceal abscess, a fistula between the appendiceal abscess and the cecum; acute bilateral salpingo-oophoritis and pelvic peritonitis.

In this case acute appendicitis and acute pelvic inflammatory disease were present at the same time.

*Threatened Abortion.* Threatened abortion is not usually confused with acute appendicitis. It is distinguished by the history of preceding amenorrhea, the labor like character of the pain, the absence of local signs and vaginal bleeding. It must be emphasized again, however, that acute appendicitis may occur in the course of an abortion and may quickly develop to an advanced stage if it is not recognized and treated.

*Ectopic Pregnancy.* Ectopic pregnancy seldom gives rise to symptoms before rupture occurs and is difficult to diagnose prior to that event though pelvic examination may reveal a tender rounded swelling on one side of the uterus or the other. In the typical case of rupture there is sudden stabbing abdominal pain associated with pallor and rapid thready pulse, lowered blood pressure and other manifestations of loss of blood including syncope and sometimes shock. Vomiting is not frequent. Inquiry into the previous menstrual history, if sufficiently detailed, will usually reveal some irregularity though not necessarily a period of amenorrhea. Vaginal examination reveals a bloody discharge, a softened cervix which is extremely tender on movement, tenderness in all the vaginal fornices and a boggy mass in the cul de sac. If the bleeding has been slight the clinical picture is less clear cut and differentiation may be difficult though cautious puncture of the cul de sac is frequently conclusive. To wait for blood studies and other laboratory tests may jeopardize the patient's life and immediate operation is best since ectopic pregnancy is an even more urgent surgical state than acute appendicitis.

A case of ectopic pregnancy reported by Plewes illustrates an unusual coincidence of diseases.

A 31 year old woman 13 years after her last pregnancy complained for 5 months of aching pain in the lower abdomen. For the first 3 months of her illness she suffered from frequency of micturition and for the last 4 months there was irregular vaginal bleeding. Shortly before she entered the hospital she was increasingly aware of a dull ache in the right lower quadrant. Operation re-

vealed bilateral pyosalpinxes a right sided abdominal pregnancy of 4 months duration and a gangrenous appendix distended with pus deeply embedded in the right pyosalpinx

Under such circumstances a definite diagnosis could scarcely be established

*Strangulation of Pelvic Cysts and Tumors* The pain produced by the strangulation of a pelvic cyst or tumor whether it arises from the ovary or the uterus is more severe and abrupt than the pain of acute appendicitis. In most cases there is some degree of shock. Dermoid cysts of the ovary are particularly likely to undergo twisting. A knowledge of the previous existence of a tumor or cyst is always suggestive particularly when localized tenderness and rigidity make it difficult to demonstrate the mass at the time of the attack. Nausea and vomiting are often present shortly after the onset but fever is a later development. If the strangulation is not interrupted gangrene will ensue as the circulation is progressively cut off. Bimanual examination is usually conclusive. When peritonitis has developed it is difficult to determine whether the primary lesion is in the appendix or in the pelvic structures. The differentiation between a strangulated pelvic cyst or tumor and acute appendicitis is not important and no time should be wasted on it since both conditions are urgently surgical.

### SURGICAL DISEASES

Not a great deal of time need be spent in the discussion of surgical states likely to be confused with acute appendicitis since most of them are as urgently surgical as is acute appendicitis.

*Perforated Peptic Ulcer (Cancer)* A perforated peptic ulcer or a perforated gastric cancer because the gastric or duodenal contents gravitate along the outer side of the colon and collect in the right iliac fossa may give rise to symptoms and signs of acute appendicitis especially if the patient is not seen until several hours have elapsed since the accident. Differentiation then must be made chiefly by the history which may or may not include the previous symptoms suggestive of the primary condition. The initial pain of rupture of a gastric or duodenal perforation is far sharper than that of acute appendicitis. Examination of the abdomen immediately after the rupture reveals board like rigidity chiefly above the umbilicus. There is definite tenderness in the right hypochondrium and pain is felt on the top of the right shoulder over the right acromioclavicular joint. Fever is not



usually present until peritonitis has set in but the pulse is rapid and the blood pressure low if the patient is not in a state of shock he is often very close to it In Cope's opinion the diagnosis of perforation of an ulcer is certain if in the absence of generalized distention obliteration of liver dullness can be demonstrated A flat interoposterior plate of the abdomen in the erect position will demonstrate the presence of air beneath the diaphragm in 80 per cent or more of all cases the proportion is even higher if the patients are examined in the right lateral position

If leakage from the perforation has been slow acute symptoms and signs are absent and such physical findings as can be demonstrated are limited to the upper abdomen Vomiting may or may not be present

*Biliary Tract Disease* Acute biliary tract disease is usually initiated by epigastric pain which is of a boring character and which if caused by stones or colic is almost intolerable and must be relieved by morphine The pain sometimes radiates to the infrascapular region (Bois sign) The initial pain tends to remain at the point of origin in all biliary tract disease and to progress upward rather than downward Vomiting frequently accompanies the pain

The maximum tenderness and rigidity are usually just below the tip of the ninth costal cartilage which is much higher than the usual point of maximum tenderness in acute appendicitis If however the gallbladder is enlarged or the liver low the maximal tenderness may be very near McBurney's point while a high inflamed appendix may present maximum tenderness in the region of the gallbladder There is usually tenderness on pressure under the ribs when the patient takes a deep breath In thin subjects the enlarged tender gallbladder can be palpated Roentgenologic examination may reveal the presence of stones

Rupture of the gallbladder is attended by the usual symptoms of ruptured viscus that is pain shock and signs of peritonitis

Biliary tract disease is not very frequent in young subjects and is most likely to appear in obese subjects and in women who have borne many children It is frequently possible to obtain a history of preceding attacks of biliary colic or a history of qualitative dyspepsia Jaundice may or may not be present it may precede the actual attack or may not appear until 24 hours after the onset

A surgeon who approves of immediate operation in acute cholecystitis will not waste much time in making the differential diagnosis A surgeon who does not approve of immediate surgery must make the

differentiation on the basis of the history of the acute attack the preceding history and the physical and roentgenologic findings. If there is any suspicion of acute appendicitis gallbladder visualization tests are contra indicated and many indeed disapprove of their use in acute cholecystitis under any circumstances.

**Acute Pancreatitis.** Acute pancreatitis presents such severe early symptoms and signs that it is unlikely to be confused with appendicitis. There is intense abdominal pain usually localized to the epigastrium but often extending to the back and the left flank. Vomiting is persistent. Abdominal palpation reveals exquisite tenderness and some resistance but no rigidity. Eventually a mass becomes palpable. The pulse is rapid and of small volume the skin is often gray and cyanotic and the patient is obviously in a state of collapse. A history of previous attacks of biliary colic is significant. The serum amylase test is diagnostic and requires only a few minutes to perform. Pancreatitis like mesenteric vascular occlusion seldom occurs in patients under 40 years of age. The hemorrhagic variety is a surgical emergency.

**Intestinal Obstruction.** Acute intestinal obstruction may begin acutely or with mild pain most often about the umbilicus. In either instance it is suggestive of acute appendicitis. There is sometimes a story of constipation obstipation or alternating diarrhea and constipation. Vomiting may be present or absent in the early stages. Later it may be persistent continuous and sometimes projectile. Fever is not an early manifestation.

In this connection might be mentioned the case reported by Moloney of acute appendicitis simulating gallstone ileus. The symptoms were compatible with intestinal obstruction and flat plate of the abdomen revealed what was thought to be an impacted gallstone. At operation however a fecalith measuring 13 by 75 by 75 inches was found in a gangrenous appendix.

Localization does not occur in intestinal obstruction and rigidity is a late sign. Distention is likely to be most marked over the actual site of occlusion though it may be generalized. In volvulus it is often enormous. Peristalsis in the early stages of obstruction is always active and is sometimes clearly visible the intestinal coils being traced on the abdomen in a ladder effect. In early cases auscultation reveals a noisy abdomen. In later cases the abdomen is ominously silent. Free fluid is frequently demonstrable on examination and roentgenologic examination usually reveals fluid levels.

The great danger of confusing intestinal obstruction with acute appendicitis is that operation may be delayed if the diagnosis of intes-

tinal obstruction is made, in order to practice decompression by the Wingensteen method with the Miller Abbott tube. If the condition is really acute appendicitis irreparable time may thus be lost as several cases at Charity Hospital of Louisiana at New Orleans clearly show. It must also not be forgotten that a perforated appendix may be the underlying cause of an intestinal obstruction (p. 272).

Intussusception in children is only occasionally confused with acute appendicitis as the picture is usually characteristic. A perfectly well child suddenly screams with pain and perhaps vomits. Soon afterward bloody mucus appears in the stool and within a few hours a sausage shaped mass is distinctly palpable. The appendix sometimes takes part in the intussusception but whether it does or does not, the condition is urgently surgical and no time need be spent in refinements of diagnosis.

*Mesenteric Vascular Occlusion* Mesenteric vascular occlusion can be conceived of as a vascular species of intestinal obstruction. In the usual case the symptoms include sudden agonizing pain most often paroxysmal in the mid abdomen or over the entire abdomen vomiting coincident with the pain prompt distention which may reach an extreme degree and usually complete absence of muscle spasm. The temperature is subnormal the pulse is weak and rapid and the blood pressure is low. The patient usually presents the appearance of shock. If the bowels move which is not usual the stool contains blood. The patient is much more acutely and gravely ill than a patient with acute appendicitis and confusion of the two conditions can occur only in atypical cases. Mesenteric vascular occlusion is urgently surgical.

*Diverticulitis* Diverticulitis of the colon is most often confused with acute appendicitis when the appendix is on the left side or when the tip extends across to the left. The symptoms and signs are usually limited to the left lower abdomen because the disease is most often limited to the sigmoid colon. If the colon is displaced the differentiation from acute appendicitis may be impossible especially in older patients. A preceding history of intestinal disturbances is significant particularly a history of alternating diarrhea and constipation or of the passage of mucus and blood. The initial pain of diverticulitis is likely to be hypogastric as compared with the usual initial pain of acute appendicitis which is likely to be in the epigastrium or about the umbilicus.

Solitary diverticulitis of the cecum is apparently impossible to differentiate from acute appendicitis on the basis of any symptoms or signs. Up to 1945 according to Gatewood 47 cases had been recorded.

including the 2 cases he himself was reporting and in these the pre-operative diagnosis had been made correctly in only one instance. Most often appendicitis was thought to be present though the pathologic stage suspected varied from chronic through abscess. Symptoms and signs included abdominal pain, nausea and vomiting, diarrhea, chills, rebound tenderness and rectal tenderness. A mass was frequently present. Differentiation apparently can be made only at operation which is required in cecal diverticulitis as well as in acute appendicitis. Gatewood's point is well taken that when a normal appendix is found at operation performed on the diagnosis of acute appendicitis a search for diverticulitis of the cecum should always be carried out.

*Meckel's Diverticulum* Meckel's diverticulum which is usually located in the small intestine 2 or 3 feet from the ileocecal junction is chiefly found in children. The symptoms are difficult if not impossible to differentiate from those of acute appendicitis on which diagnosis operation is usually performed. Local tenderness is frequently at the level of the umbilicus and therefore is higher than the typical tenderness of acute appendicitis. The disease is inflammatory and may go on to gangrene and rupture. Since operation is usually necessary the differential diagnosis is of academic interest only. Meckel's diverticulum should always be looked for in any operation for acute appendicitis in which exploration is not contra-indicated.

*Regional Ileitis* Only the acute variety of regional ileitis (Crohn's group 1) is likely to be confused with acute appendicitis and it is generally agreed that pre-operative differentiation is practically impossible. Pain and tenderness in the right lower quadrant, fever and leukocytosis are characteristic of both diseases. If the abdomen is opened the appendix is likely to be found involved by contiguity in the ileal inflammation but there is no mucosal involvement.

*Carcinoma of the Right Half of the Colon* Carcinoma of the right half of the colon (including the cecum) is often associated with recurrent attacks characterized by severe pain, vomiting, tenderness, localized distention and sometimes rigidity. A tumor mass resembling an appendiceal abscess may be palpable. It is also possible as happened in 9 surgical and 2 (fatal) nonsurgical cases at the New Orleans Charity Hospital that acute appendicitis may be mistaken for carcinoma of the cecum. Since patients with intestinal malignancy are elaborately prepared for surgery by purgation among other measures the importance is clear of differentiating the disease from acute appendicitis in which prompt operation is required and purgation is

disastrous The severe anemia often present in carcinoma of the right half of the colon is a helpful differentiating point

Confusion between the two conditions is not likely to arise except in older subjects in whom appendicitis is atypical and in whom fever and other signs of inflammation are often less marked than in younger subjects The possible coincidence of carcinoma of the intestine and acute appendicitis was pointed out by Bartlett and Miller who observed 2 cases personally and collected 6 others from the literature the age range in the 8 cases was from 32 to 70 years A somewhat similar instance in a considerably younger subject was observed at the New Orleans Charity Hospital

*Case 16* A 13 year old Negro boy complained for 6 days of moderately severe cramping pain in the lower abdomen For 3 days he had vomited everything taken by mouth Except for a small stool shortly before he entered the hospital he had had no bowel movement for 4 days There was a history of occasional indigestion over an indefinite period of time The temperature was 100.2 F the pulse 114 and the respiration 26 The patient was slightly emaciated and somewhat dehydrated but did not seem seriously ill The abdomen was slightly distended and deep palpation elicited moderate tenderness in both lower quadrants A loop of bowel palpable in the right lower quadrant intermittently became a large firm mass with peristalsis Rebound tenderness was questionable Rectal examination revealed tenderness of the posterior abdominal wall A soft mass felt rather high was suggestive of redundant mucosa but proctoscopic examination revealed nothing abnormal in the 7 inch area examined A flat plate of the abdomen revealed two fluid levels The white blood cell count was 10 900 and the red blood cell count 3 500 000 per cu mm

Although no history of parasitism could be secured intestinal obstruction due to that cause was suspected and operation was undertaken under ether and ether anesthesia through a right rectus incision The small intestine was greatly distended and the cecum slightly distended In the colon 6 cm distal to the hepatic flexure was a napkin ring constriction clinically suggestive of adenocarcinoma The appendix was thickened reddened and retrocecal Appendectomy and ileotransverse colostomy were performed Examination of the appendix in the laboratory revealed that it was acutely inflamed edematous and diffusely hemorrhagic it was filled with purulent fluid It contained a fecalith and cysts of *E. histolytica* were identified Following a smooth recovery from the first operation the patient was submitted to resection of the right half of the transverse colon from which he also made a smooth recovery The neoplasm was reported as adenocarcinoma

*Primary Torsion of the Omentum* Up to 1945 according to Altemeier and Holzer only 70 cases of primary torsion of the omentum had

been put on record in the literature including 6 which they themselves were reporting. In every instance in their personal series as in most of the reported cases the pre-operative diagnosis was acute appendicitis and the two conditions seem clinically indistinguishable except that the progression of symptoms is perhaps less rapid in omental torsion than in acute appendicitis. The differentiation is of no practical diagnostic importance since both conditions are surgical.

Harris Diller and Marcus observed 2 cases of hemorrhagic infarction of the greater omentum both of which simulated acute appendicitis. They note that in this condition torsion of the involved portion of the omentum may or may not occur.

### CONCLUSIONS

This chapter on differential diagnosis like the preceding chapter on the clinical picture and the diagnosis of acute appendicitis may seem inconclusive. No apology is offered. Differential diagnosis is possible in the great majority of cases of acute appendicitis if the proper efforts are made to establish it. If after such efforts have been conscientiously made and after all possibilities have been evaluated the diagnosis still cannot be made then abdominal exploration becomes a justified diagnostic procedure. It is not possible and it would not be safe or wise to lay down strict points of differentiation for many of the myriad diseases which can mimic acute appendicitis or which acute appendicitis can mimic. The endeavor in this chapter has been merely to suggest important points for consideration and to suggest also the range of diagnostic possibilities in acute appendicitis in which the clinical picture is atypical. That group of cases as has been pointed out includes from a quarter to a half of all cases. If these facts are borne in mind however not many cases of acute appendicitis will be overlooked even if they are atypical.

## (IX)

### *The Complications of Acute Appendicitis*

#### I PERITONITIS, PRIMARY PERI-APPENDICEAL ABSCESS, RESIDUAL ABSCESS, AND PYLEPHLEBITIS

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##### PERITONITIS

##### INCIDENCE

The reported incidence of peritonitis following rupture of the appendix shows wide variations. In the various series which made up the more than 10 000 cases of acute appendicitis collected from the literature by Ochsner Gage and Garside in 1930 it ranged from 6.5 to 77.6 per cent depending first of all upon whether the base figures included all varieties of acute appendicitis or only cases of rupture of the appendix. The incidence was 17.64 per cent (3 422 cases) in the 19 398 cases of acute appendicitis included in the 1940 report of the Commission on Acute Appendicitis Mortality of the Medical Society of the State of Pennsylvania which adopted as its definition of peritonitis inflammation of the peritoneum following perforation of the appendix. 2 312 cases (68.4 per cent of all cases of peritonitis) were of the spreading type. In the 1945 report of the same Commission which included 23 681 cases of acute appendicitis the incidence of peritonitis was 9 per cent (2 143 cases). 566 cases (26.4 per cent of all cases of peritonitis) were of the spreading variety.

In the 6 441 cases of acute appendicitis analyzed in 11 separate studies at the New Orleans Charity Hospital over the 15½ year period ending 31 December 1945 there were 1 698 stated cases of definite peritoneal involvement (25.9 per cent). 534 (32 per cent of all peritonitis cases) were of the spreading variety. These figures include all cases of peritonitis caused by acute appendicitis and they probably should be considerably higher since the records in the earlier years of the study were frequently of doubtful accuracy. There has been a steady decrease in the incidence of peritonitis at Charity Hospital as the studies have progressed undoubtedly as the result of earlier diagnosis, prompt treatment.

ment and prompt exploration in doubtful cases. It is obvious however that much remains to be done in the way of public education when an essentially simple and readily curable disease such as acute appendicitis is still complicated by peritonitis in approximately 17 per cent of all cases which was the situation in the last series of cases (1941-5) studied from the New Orleans Charity Hospital.

#### CLINICAL PICTURE

*Symptoms* The symptoms of peritonitis are necessarily modified by whether the disease (1) is part of a progressive process which has developed before the patient is seen by the physician (2) develops in the course of expectant treatment undertaken in the mistaken belief that the appendix has already ruptured and localization is occurring (3) is a postoperative development or (4) is observed in a neglected case seen for the first time in its terminal stages.

Cope has divided the symptoms and signs of peritonitis into two groups reflex which occur early and toxic which occur late. Reflex symptoms and signs are pain vomiting anxious facies abdominal muscular rigidity superficial hyperesthesia collapse and temperature alterations. Toxic symptoms are collapse temperature alterations distention intestinal paresis and general toxemia.

Reflex symptoms appear more promptly when the disease process affects the demonstrative part of the peritoneum. They may be considerably delayed when it affects the nondemonstrative part. The distinction is based on whether or not the cerebrospinal nerve supply to the affected part is free or scanty. The anterior and lateral parts of the abdomen are lined by peritoneum which is well supplied by somatic nerves so that the reflexes are brisk. The pelvis and median portion of the posterior abdominal wall have a scanty nerve supply and irritation in consequence causes minimal reflex symptoms. Furthermore early reflex symptoms are more likely to be definite in young persons whose reflex arcs are normally more sensitive and may be relatively insignificant in elderly and debilitated persons in whom the stage of toxemia may come on almost insidiously because reflex symptoms have been so mild that they have attracted little attention.

There is an inverse relationship between toxic and reflex symptoms since severe toxemia diminishes the sensibility of the reflex arc. When the pelvic or other nondemonstrative parts of the parietal peritoneum are primarily affected reflex symptoms may be minimal throughout since the onset of toxemia diminishes the reflexes from the demonstrative





volume weak and thready. When the temperature begins to fall and the pulse begins to rise so that the lines meet and cross on the chart in the so called cross of death (fig. 47) recovery is not to be expected.

Collapse which is chiefly evident in impairment of the circulation may occur early or late in peritonitis. It is most likely to be manifest clinically in cases in which the onset is clear cut and in which the demonstrative areas of the peritoneum are involved. Otherwise collapse and the toxic stage of peritonitis can scarcely be differentiated from each other.

Late collapse is associated with toxemia and is far more serious than collapse in the early stages when the process is usually reversible under proper management. As the disease progresses throughout the abdomen more and more coils of intestine become paralyzed and distended. Ileus is then a pathologic and no longer a local protective process; the intestinal contents stagnate and the patient as a result becomes extremely toxic. Obstructive or regurgitant vomiting occurs. The temperature falls to normal or subnormal and the pulse is small and very rapid. The patient may be listless and apathetic, entirely oblivious to his surroundings or actually comatose. Less often he is in a state of euphoria which is always a bad sign in fact if his status improves in his own opinion as it becomes worse in the opinion of his physician, death can almost surely be expected.

*Physical Findings.* The patient with peritonitis is likely to lie motionless in bed with the knees pulled up to relieve tension on the abdominal muscles. The facies is drawn and pallid but the typical hippocratic facies with sunken eyes, pinched nose and apprehensive stare is not present in the early stages unless collapse has occurred. When it is present as Cope has succinctly put it it tells the physician at a glance much that he ought to know but is sorry to learn.

*Tenderness* is the most constant physical finding. It may be localized or most marked over the initial focus or generalized. If it is not immediately apparent it can be elicited by deep pressure unless collapse has occurred early or unless the patient is seen so late that toxemia has dulled the sensorium. This had occurred in a surprising number of the cases studied at Charity Hospital in both the surgical and nonsurgical groups particularly among the older subjects.

*Muscular rigidity* which is always present in early peritonitis if the demonstrative area of the peritoneum is affected is localized or generalized according to the extent of the involvement. The initial picture of peritonitis may be confused because extreme muscular rigidity may

prevent the transmission of applied pressure to an underlying inflamed area (Cope)

When a pelvic appendix has ruptured particularly a deep lying pelvic appendix tenderness and rigidity are often not immediately demonstrable. When a more superficially located appendix has ruptured tenderness and rigidity of the whole lower abdominal wall may be at once apparent and the symptoms are likely to be proportionately more marked. In such cases abdominal distention is also an early sign.

*Localizing Peritonitis* The symptoms of localizing peritonitis are little more than those of the initial lesion that is local pain and tenderness muscle spasm acceleration of the temperature and pulse rate and an increase in leukocytosis. The temperature and pulse rate may be somewhat higher than in the initial disease. The physical findings may also be little more than those of the initial lesion. In some instances a mass is felt which may be found to consist of the appendix, the intestine and the omentum. The interpretation of abdominal masses in the appendiceal region however often assumes the aspects of a game of chance and what is taken to be a ruptured appendix with localized peritonitis may prove to be an intact appendix, delay in the removal of which might have been attended with serious consequences (p. 204).

*Postoperative Peritonitis* Peritonitis which was not present before operation may develop afterward for a variety of reasons such as contamination of the peritoneum by technical errors at operation failure to drain the peritoneal cavity when drainage is indicated contamination of the peritoneum by transperitoneal drainage of abscesses or according to Bower the performance of ill timed operations by which the formation of adhesions is interfered with and a local infection is converted into a general one. Opponents of conservative therapy in ruptured appendicitis naturally do not accept the latter cause provided that operation has been performed properly.

Peritonitis which develops after operation does not differ greatly from the type which has already been described. The patient does not improve as he should with the removal of the infective focus. Pain persists usually diffuse and dull rather than sharp and localized. Vomiting is the rule. Tenderness and rigidity are usually present and are associated with distention and tympanites as the intestines become progressively more involved. The temperature is likely to be persistently high and the pulse is rapid. The patient looks and is very ill.

In other instances the patient improves after operation for a period of 48 or 72 hours or even longer. Then the earlier symptoms especially

pain nausea and vomiting recur the temperature and pulse are elevated and the clinical picture is that of the usual type of peritonitis

#### ILEUS AND MECHANICAL INTESTINAL OBSTRUCTION

*Pathologic Process* Ileus is a term borrowed by usage though intestinal incompetence as suggested by Long in 1924 is more accurate and more descriptive

Ileus of mild degree follows almost all operations that involve manipulation of the intestines. It is invariably present when the procedure has been traumatic since it represents the response of the intestine to any sort of abnormal stimulation whether inflammatory bacterial or mechanical. Extreme degrees are most often found in appendicitis with rupture and peritoneal involvement though ileus may be a serious complication in gangrenous appendicitis or even in suppurative or simple acute appendicitis.

Simpson from personal observation in a small number of patients concluded that ileus is particularly likely to occur after operation in cases in which the mesentery of the terminal ileum is so shortened by scar tissue or thickening as to cause torsion and lateral rotation of the ileum. The changes are presumably the result of recurrent inflammation of the appendix or may be the result of inflammation of the lymphatic vessels draining the appendiceal region.

Ileus in the early stages of acute appendicitis and peritonitis is a localized and protective process since it brings about a decrease of peristaltic activity in the region of the diseased organ and thus provides the state of physiologic rest desirable in the treatment of any disease. As it involves more and more of the intestine however the process becomes pathologic and differs in effect from mechanical obstruction only in that there is no single point of blockage.

The bowel normally contains a certain amount of gas which since it is controlled by the normal tonus of and the normal circulation in the intestinal wall does not reach excessive proportions. When intestinal tonus is impaired as happens to some degree in all intra abdominal operations or when it is relatively or absolutely destroyed as frequently happens in advanced acute appendicitis the amount of gas is not controlled and ballooning of the bowel occurs. The amount is increased by swallowed air failure of absorption of gases from the intestinal tract as the result of circulatory impairment and diffusion of dissolved gases especially nitrogen from the blood stream into the intestine. Digestive juices and toxic products also accumulate within the

*intestine* As a result of these various phenomena the intraluminal pressure becomes increasingly higher

The exact cause of ileus is not clearly understood though there is no particular difficulty in explaining it in elderly and debilitated patients and in patients in whom dehydration has not been corrected Another obvious cause is oral feeding too soon after operation before normal peristalsis has returned It has been suggested that some nervous factor such as excessive irritation or excessive stimulation of the splanchnic nerves may be responsible and disturbance of the chemical equilibrium has also been suggested though it would seem more reasonable to regard chemical changes as the result of peritonitis and ileus rather than as the cause of ileus

If ileus continues unchecked the blood supply of the intestine becomes impaired as a result of increased intraluminal pressure the intestinal wall becomes thinner and thinner and free fluid appears in the peritoneal cavity as the result of permeability changes and transudation Gangrene of the intestine is a possible outcome in any case in which excessive distention continues unchecked it is always to be suspected when the peritoneal fluid is bloodstained When ischemia of the bowel wall develops even if gangrene is not yet present toxic products are readily absorbed from the peritoneum and the clinical state of toxemia comes on This is not the place to discuss whether or not there is a true toxin of intestinal obstruction and ileus Whether there is or is not there is a true toxemia associated with intestinal obstruction and ileus and it is responsible for a large proportion of the deaths in these conditions

The pathologic effects of ileus accompanied by marked distention are for all practical purposes similar to those of mechanical intestinal obstruction even to disturbances of the chemical balance (p 309) The elevation of the diaphragm which occurs when extreme distention is present may embarrass both respiration and cardiac action and may cause a compression atelectasis and subsequent pneumonia

The ileus which normally follows all surgical procedures is frequently so slight as to give rise to no clinical manifestations A patient with a pathologic degree of ileus is likely to complain of considerable nausea followed after a lapse of time by vomiting increasing abdominal distention and great abdominal discomfort in contrast to the colicky true pain of intestinal obstruction of mechanical origin The pulse rate is increased frequently out of proportion to the temperature elevation In late ileus auscultation reveals an ominously silent abdomen

*Mechanical Intestinal Obstruction* Mechanical intestinal obstruction

Fig 48 Flat plate of abdomen showing mechanical obstruction following appendiceal abscess. Note distended loops of small bowel and calcified fecalith in appendix (from the University of Minnesota Hospitals Minneapolis)



after appendectomy or in the course of complicated acute appendicitis which is being treated conservatively does not differ from the type of mechanical obstruction observed in other circumstances. It is chiefly due to the formation of adhesions which are part of the protective process. Although they are light and filmy in contrast to the firm adhesive bands seen in obstruction which occurs weeks or months after operation they are quite capable of causing intestinal occlusion. Furthermore even slight adhesions may cause dangerous angulations in a peristaltic bowel which is unduly heavy because of the weight of its contents. Such angulations may also occur in the absence of adhesions. Incorporation of the intestine in an appendiceal abscess is a rather frequent cause of ileus and a less frequent cause of mechanical obstruction (fig 48). This is another instance in which the pathologic process develops from the natural protective process. Any portion of the intestinal tract may be incorporated in an appendiceal abscess but the ileum and cecum are most frequently affected.

Although mechanical intestinal obstruction is a theoretic possibility in any case of complicated acute appendicitis it does not occur very often which is fortunate for it is highly fatal. It was observed in only 23 of the 6441 surgical cases studied from the New Orleans Charity Hospital but it was fatal in 14 of these 60.9 per cent. Release of the obstruction was carried out in 12 cases 5 of which were fatal probably because the differential diagnosis between ileus and obstruction was delayed too long for surgery to be of any avail.



Fig 49 Flat plate of abdomen showing adynamic ileus in peritonitis of appendiceal origin. Note distended loops of bowel.



Fig 50 Roentgenogram taken in upright position in case of mechanical intestinal obstruction with appendico ileal fistula following rupture of appendix into ileum. Note fluid levels.

**Diagnosis** The distinction between ileus and mechanical obstruction is extremely important because the latter usually must be treated surgically whereas in the former condition surgery not only does no good but actually aggravates the process. Diagnosis however is not always simple. Mechanical intestinal obstruction which begins abruptly is usually fairly easy to diagnose but obstruction arising on the basis of ileus which is most often the situation in peritonitis following rupture of the appendix presents serious problems. The colicky pain is interpreted as returning peristalsis if only because that is what the surgeon hopes is happening and while he waits to confirm his hopes the patient's condition if true obstruction is present becomes progressively worse. For this reason it has been wisely suggested that a surgeon who is confronted with such a problem should immediately call in a consultant who is likely to have a more dispassionate outlook even though he perhaps possesses no greater diagnostic acumen.

In both ileus and mechanical intestinal obstruction roentgenologic examination in the upright position with a flat plate will show numerous fluid levels due to the presence of gas and fluid in the intestinal coils. The diagnosis of intestinal incompetence is thus confirmed but an x-ray unfortunately usually makes no clear-cut distinction between ileus and

mechanical obstruction (figs 49 50) If auscultation does not make clear whether the sounds within the abdomen are due to returning peristalsis after ileus or are due to hyperperistalsis as the obstructed bowel endeavors to empty its contents and if a brief period of decompression (p 300) and replacement therapy (p 307) does not relieve the symptoms the patient's best interests are usually served by an exploratory operation

#### DISTURBANCES OF THE FLUID AND CHEMICAL BALANCE

As a matter of convenience disturbances of the fluid and chemical balance associated with peritonitis are discussed in the chapter on preoperative and postoperative care (p 307) Here it need only be stated that such disturbances are constant in all cases of advanced appendiceal disease that their degree generally speaking is related to the stage of the pathologic process and the duration of the illness and that the result of attempts at correction depends upon whether or not the changes have reached irreversible proportions

#### PRIMARY PERI APPENDICEAL ABSCESS

The final outcome of a ruptured appendix in which the pathologic process has localized or of a diffuse peritonitis which has shown the same localizing tendency is abscess formation Whether this is altogether the happy outcome which some observers seem to believe it to be is a matter of considerable doubt (p 206) Whether localizing tendencies are evident in all cases of rupture of the appendix as some observers claim is also a matter of doubt Since however these considerations are intimately concerned with the propriety of conservative therapy in advanced acute appendicitis they are more conveniently discussed under that heading

The mortality of peri appendiceal abscess at the New Orleans Charity Hospital (59 of 449 surgical cases 13.14 per cent plus 12 fatal cases in which operation was not done) does not support the idea even after all allowances are made for improper selection of cases for surgery and improper application of conservative therapy that abscess as Love expressed it is the most fortuitous outcome of the ruptured case The figures (fig 71 p 337) suggest instead that abscess is a desirable termination of complicated acute appendicitis only as contrasted to spreading peritonitis

*Incidence* The incidence of appendiceal abscess varies widely Hicken and Carlquist in contrast to their own incidence of 10 per cent



in 523 cases of acute appendicitis cited Arnheim and Neuhof's incidence of 18.8 per cent Guerry ■ of 18 per cent and Bowers of 15 to 20 per cent They explained their own relatively low incidence by the fact that their patients were seen chiefly in private practice and therefore did not represent the type of delayed case observed in many other reported series In the more than 10 000 cases of acute appendicitis of various types collected from the literature by Ochsner, Gage and Garside the incidence in separate series varied from 5.2 to 67 per cent In the 6 441 cases of acute appendicitis studied at the New Orleans Charity Hospital (fig 70, p 337), there were 449 peri appendiceal abscesses This number represents 6.97 per cent of all cases of acute appendicitis and 26.45 per cent of all cases of rupture (1 248 cases)

*Clinical Picture* The symptoms and signs of primary peri appendiceal abscess are widely varied They depend upon the location of the abscess (fig 40 p 100) the time at which the patient is seen and his individual resistance as well as his individual threshold of pain and discomfort An occasional patient with a well developed abscess may have been ambulatory or almost ambulatory throughout most of his illness and may stoutly deny any but the most casual symptoms and signs

The symptoms of appendiceal abscess frequently do not differ from the symptoms of localizing peritonitis and often do not differ greatly from the symptoms of uncomplicated acute appendicitis The preceding course may have been mild or stormy and the symptoms may be those of the classical disease or may be unusual and bizarre just as they may be in any other kind of appendicitis The temperature may be very high or only moderately elevated and the same holds for the pulse rate while leukocytosis is not in any way typical

In a clear cut case of appendiceal abscess it is often possible to demonstrate a right sided mass though the mere presence of ■ mass unless fluctuation can also be demonstrated does not necessarily prove that one is dealing with an abscess Collins for instance, among ■ 712 cases of acute appendicitis found 379 masses which proved to be acutely inflamed perforated appendices without abscess formation matted coils of ileum or omental masses If delay had been practiced in these cases he noted in the belief that an abscess was present the outcome could easily have been fatal

Abscesses in certain locations cause certain symptoms and signs Thus an abscess overlying the ureter will cause urinary symptoms A pelvic abscess characteristically gives rise to rectal tenesmus and diarrhea and sometimes to urinary disturbances Occasionally it causes

edema of the scrotum. An abscess intimately connected with coils of intestine may give rise to symptoms varying from those of gastroenteritis to those of mechanical intestinal obstruction. Hicken and Carlquist's discussion of signs and symptoms on the basis of the location of the periappendiceal abscess is ingenious but it is doubtful that even experienced physicians should rely upon their diagnostic acumen to the extent of diagnosing not only the existence of an abscess but its exact location.

# THE DELAYED (EXPECTANT CONSERVATIVE) TREATMENT OF APPENDICEAL PERITONITIS AND PERI APPENDICEAL ABSCESS

There is almost no argument concerning the urgency of the treatment of acute appendicitis in its early stages (though some discussion about how long early is) and there is general agreement that the proper treatment is prompt appendectomy. When however the patient seeks medical consultation late or when the diagnosis for any reason has been delayed and peritonitis and abscess formation have occurred there is far less agreement about the best course. The pendulum swings from one side of the question to the other and perhaps the fairest method of presenting the case is to outline the arguments for conservative therapy in these circumstances and to set against them the arguments for immediate surgery for acute appendicitis in all stages.

## EXPECTANT TREATMENT

Bower perhaps the outstanding advocate in the United States of conservatism in advanced acute appendicitis has reportedly stated that one of the reasons for the reduced mortality of the disease in Philadelphia is the expectant treatment of appendiceal peritonitis. Many of whom I am one think it far more reasonable to attribute the remarkable improvement in that city to the increasing proportion of unruptured cases which come to operation and the increasing proportion of cases in which early operation is done both of which achievements are directly attributable to Bower's own efforts.

On the basis of experimental studies by his own group and by other groups of observers Bower argues that expectant treatment is justified in advanced acute appendicitis because (1) it takes time for immunity to develop in appendiceal peritonitis (2) the end result of an antigen cell reaction is essential for the patient's protection and (3) the desired end result of the protective process

that is a localized collection of pus will not come to pass if natural processes are interrupted by local surgical interference. If the localizing process is not disturbed a localized abscess will develop and the patient will be protected by a fully developed immunity. When this has happened the tissues that form the abscess wall and those contiguous to it possess a local immunity and in most instances antitoxin is circulating in the blood stream. When a localized abscess has not yet developed and the process is still a *localizing* peritonitis the local and general immunity is undeveloped as are the local walling off structures.

Spreading peritonitis in Bowers opinion is frequently induced by surgical interference with the localizing process. The surgeon who pushes aside and separates adherent loops of intestine and omentum is injecting into the blood and lymph stream a dose of antigen which may be lethal. According to Bowers reasoning patients receive doses of this antigen first through the serosa intact appendix and second through the perforated appendix. A third dose received as the result of the search for and the removal of a leaking appendix may be lethal. No matter how carefully operation is done protective adhesions are broken down and a localizing process can be converted into a spreading one. The theory advanced by Bower is thus based on the concept that operation is dangerous in the so called negative phase of infection. It is directly opposed to the concept that early removal of a focus of infection is the only rational procedure.

#### IMMEDIATE OPERATION

Elman and Stone may be cited as representative of the school (to which I also belong) that argues that the existence of peritonitis does not justify delay in removal of the causative perforative lesion in the appendix *exclusive of the necessary delay for the rehabilitation of the poor risk patient*. Elman's position is that all the surgeon need know is whether a patient has acute appendicitis and whether he is in sufficiently good condition to withstand surgery. Delay is unjustified even in localizing lesions because (1) There is no certainty that an abscess will remain localized and disappear and definite danger that it will rupture and spread infection throughout the peritoneal cavity (2) It is impossible to be certain whether the localized mass in the right side is really an abscess or is merely an inflamed appendix surrounded by intestine and omentum (3) The diagnosis of rupture of the appendix is notoriously inaccurate.

Stone's arguments are much the same (1) The diagnosis of acute

appendicitis is uncertain (2) It is against all reason to believe that a properly conducted operation is as harmful as permitting the continued entrance of infection from a perforated viscus into the peritoneal cavity (3) The effect of teaching the delayed treatment of certain complications of acute appendicitis has had a bad effect upon both profession and laity and has led some at least to believe that this method is possible and wise in any case of acute appendicitis Stone added that in his work as a member of the examining committee of the American Board of Surgery he had witnessed the confusion caused in the minds of physicians by the teaching and practice of conservative therapy in complicated acute appendicitis and could appreciate its harm The late Mont Reid had testified to the same confusion

The assumption on which the expectant treatment of complicated acute appendicitis is based is that the appendix has ruptured and peritonitis is present with or without abscess formation This is not a matter, however as has already been emphasized about which the most expert diagnostician can be positive Arkush and Kosky for instance studying the cases at the Santa Monica Hospital over a 10 year period from the standpoint of diagnosis found that 95 per cent had been correctly diagnosed as acute appendicitis before operation but also found that the appendix was unruptured at operation in 48 per cent of the cases in which a pre operative diagnosis of rupture had been made Murphy writing in 1904 emphasized the same possibilities of error It was formerly his custom he wrote to predict the pathologic process before he opened the abdomen and he had an excellent average of accuracy But he also had some rude awakenings He therefore changed his practice and merely said to the physicians about the operating table We do know from the clinical history the symptoms and physical signs that this is a case of appendicitis We do not know what the tendency of the pathologic process in this individual case is whether it be toward security and repair or toward destruction and danger That is about as far as even the most experienced of surgeons can safely go

Statistical evidence in favor of conservative treatment versus immediate surgery must be interpreted with caution The statistics for immediate operation necessarily include all types of cases The statistics of expectant treatment include only the cases selected for that type of treatment and the more experienced the surgeon who employs the method the higher is the degree of selection Moreover as has been pointed out many times most of the series in which excellent results of conservative treatment are reported include a number of patients who

were never very sick. Acute appendicitis is always a potentially serious disease but all patients with the disease are not always very sick.

Moreover as Stone and others have pointed out an analysis of the mortality of acute appendicitis should include all deaths from the disease. If conservative treatment is the wisest treatment for ruptured appendicitis and peritonitis it would seem that even without the employment of adjuvant therapy—which is all that expectant treatment amounts to—a number of patients with complicated disease should survive. Perhaps some do. But many die. Reid and his associates in 1936 called attention to the fact that in Cincinnati over a previous 7 year period 300 persons had died of appendicitis outside of hospitals and added that the therapeutic road of conservatism was strewn with the corpses of those who might have been saved by surgery. All public records reveal similar findings. Over the 15½ year period at the New Orleans Charity Hospital during which 320 deaths occurred after operation for acute appendicitis there were 153 fatal cases in which operation was not done. In 61 of this group the diagnosis was not made antemortem though in many all the components of expectant treatment were employed during the period of observation. Some of the remaining patients were moribund or were too ill for operation before rehabilitation which was not successfully accomplished. In 56 instances however conservative therapy was the deliberate choice of the surgeon. To these 56 deaths should properly be added 2 others classified in the surgical group in which the abdomen was opened but nothing was done when it was found that an appendiceal abscess was present.

The conservative treatment of complicated acute appendicitis over and above whether or not the patient lives or dies is by no means free from risk. It has already been mentioned that rupture of an abscess under expectant treatment is a possibility which must not be discounted. At least 4 such accidents all fatal occurred at the New Orleans Charity Hospital in the period covered by these studies. In 2 instances the rupture apparently occurring when the patients merely turned over in bed. In 2 of the 4 cases death occurred almost instantly as it did in the 2 similar cases reported by Grey Turner and the 2 reported by Vale. Rupture of an appendiceal abscess into the peritoneal cavity occurred in 1 of Bailey's 75 cases in which expectant treatment was practiced but the patient was on the threshold of the operating theater and prompt operation saved his life.

In at least 3 recognized cases in the Charity Hospital series, 1 of which was fatal rupture of a presumably unruptured appendix occurred in the hospital while the patients were under expectant treatment. In

2 other cases death occurred from hemorrhage in the course of expectant treatment. An instance of this sort follows.

*Case 17.* A colored man 34 years of age had suffered from pain above the umbilicus for 48 hours. It had been aggravated by a dose of salts taken on his own initiative and by a dose of castor oil administered by his physician who said that he did not have appendicitis. The temperature was 101.6° F., the pulse rate 118, and the respiratory rate 30. Physical examination revealed generalized abdominal tenderness and rigidity more marked in the right lower quadrant where a palpable mass could be made out.

The admission diagnosis was ruptured gallbladder versus ruptured peptic ulcer versus ruptured appendicitis. Conservative treatment including transfusion was instituted. On the ninth day tenderness was demonstrated over the ninth rib on the right side and a diagnosis of liver abscess was entertained. Rectal examination was negative. On the eleventh day the patient developed a cough and diarrhea. Several hours later he passed 250 cc. of bright red blood per rectum and went into shock. A medical consultant then made a diagnosis of typhoid fever.

The patient died the following day and postmortem examination revealed massive hemorrhage from the mesenteric vessels secondary to gangrene of the cecum following rupture and gangrene of the appendix. A peri appendiceal abscess was present.

Even admitting the good results that various experienced surgeons have been able to show for expectant treatment in advanced acute appendicitis there is full justification for prompt operation in all cases regardless of the stage of the disease because (1) Results by this plan are as good as if not better than the results of conservative therapy, (2) There is always the risk when expectant treatment is generally practiced that it will be applied incorrectly or that it will be used whether deliberately or on an erroneous diagnosis in cases of acute appendicitis in which rupture has not occurred.

Fitz's commentary on expectant treatment is interesting in this connection. "The presence of a general peritonitis," he wrote, "does not contraindicate operation." After describing several instances from the literature in which operation was performed under such circumstances he added:

If the encysted peritonitis becomes general death has heretofore been almost inevitable. It is thus obvious that if laparotomy was successful in two out of three cases [cited from the literature] where a secondary general peritonitis was present there is more than a chance of recovery by its use even in hitherto necessarily fatal cases. But it should be employed only when suitable and not as a last resort when patients are moribund.

This is still sound advice. It seems fair to say that conservative treatment if it is used at all is applicable to only a very small proportion of cases of advanced acute appendicitis and that it should be used in them only on special indications by experienced surgeons, and according to a strict routine (p. 329)

#### DELAYED REMOVAL OF THE APPENDIX

A serious consideration when nonsurgical therapy of advanced acute appendicitis is practiced or when only drainage of an abscess has been done is the patient's after history. What shall be done about the appendix? And when shall it be done?

In Love's opinion 65 per cent of all peri appendiceal abscesses will absorb spontaneously and clean appendectomy can be done within 3 months. According to Lehman and Parker the establishment of an abscess marks the end of the dangerous phase of the disease and many patients under careful treatment will never require surgery. But other series tell different stories. For that matter, 9 patients in Lehman and Parker's own series had recurrent acute attacks which in 2 instances progressed to rupture and abscess formation and 1 patient returned twice for conservative treatment of recurrent acute appendicitis before agreeing to appendectomy. In a case observed by Mayo the appendix ruptured 3 times. Sixteen of McClure and Altemeier's patients who had been treated only by drainage of abscesses had recurrent acute attacks confirmed by operation within the year. Four of Gardner's 89 conservatively treated patients returned with recurrent acute disease.

Thirty-three patients in the Charity Hospital series who were treated only by drainage of a localized abscess had recurrent acute attacks which terminated fatally in 5 instances. In the following case a fatal recurrence took place a month after operation.

*Case 18.* A colored man 33 years of age had been treated conservatively and later by drainage for peritonitis following rupture of the appendix. He returned to the hospital 4 weeks after the operation complaining of pain in the right side extending down the right leg and a mass in the right lower quadrant of the abdomen. The temperature was 99° F. the pulse rate 100 and the respiratory rate 22. The abdomen was distended, tense and rigid and a large hard mass was felt in the right lower quadrant. There was extreme tenderness in the right groin and over the mass below Poupart's ligament. The sedimentation time was 2 minutes.

It was assumed that the old appendiceal process had extended to the psoas muscle and conservative treatment including transfusion was instituted. On

the fifteenth day a retroperitoneal abscess was drained under spinal analgesia. The patient continued to run a septic temperature. Thirty days later multiple pus pockets were opened under ethylene anesthesia and counter drainage was established in the right lumbar region. The patient died on the eighth day after the second operation.

As Collier and Potter expressed it deferred surgery in complicated acute appendicitis implies that surgery is going to be done. If conservative treatment of peritonitis or simple drainage of an abscess has been practiced it should be regarded by the surgeon as preparation for appendectomy rather than as a plan of treatment in itself and the patient should fully understand the situation. To make him return for operation however is more easily said than done. Lehman and Parker admitted that only 28 per cent of their patients treated without operation and only one sixth of those treated by drainage returned as advised for interval operation. Their assumption may be correct that those who did not return for interval operation or who did not return with recurrent attacks had remained well to the date of writing because there is no other hospital in the vicinity in which they could have been treated. It is not any proof however that they will remain well.

Gardner who reported that in spite of all efforts including attempts at contact by local physicians and social service workers interval appendectomy could be done in only a little more than 50 per cent of all the patients treated conservatively in his series commented that failure in this respect was a great disadvantage of expectant treatment. Mahorner and Vincent who studied their material from this standpoint found that of 48 patients treated conservatively and dismissed without operation only 1 who did not have a recurrence returned voluntarily for surgery. One patient in the Charity Hospital series had four operations (exploration with identification of a peri appendiceal abscess but no other procedure drainage of the abscess rectal drainage of a cul de sac abscess extraperitoneal drainage of a diaphragmatic abscess) and at the end of four years still has his appendix *in situ*.

There is some difference of opinion as to when operation should be done in cases which have been treated expectantly when signs of localization first appear or as an interval procedure when the acute process has completely subsided. Mahorner and Vincent who advocated the performance of appendectomy before the patient was dismissed from the hospital after expectant treatment took the position that four weeks was the optimum maximum period of delay after subsidence of systemic evidence of the inflammatory process. They pointed out that it



is unwise to base the time of operation upon the disappearance of the mass since in at least a third of all patients masses persist for indefinitely long periods and are sometimes present as long as seven months after the acute attack. An additional reason for prompt surgery is that adhesions which are thin and easily separated after the attack become firmer and fibrotic and therefore more difficult to separate as time passes.

### RESIDUAL ABSCESSES

*Incidence and Etiology* Residual abscesses according to Ochsner Gage and Gusside occurred in from 18 to 57 per cent of the individual series comprising the more than 10 000 cases of acute appendicitis which they collected from the literature. The latter proportion is probably atypically high.

For reasons of gravity pus which forms in spreading peritonitis tends to collect in dependent pockets or basins of the peritoneum such as the pelvis the right lumbar region and the regions of the intestinal flexures (fig. 40 p. 100). Multiple abscesses and sinuses are often observed in neglected cases and may terminate if recovery ensues in thick fibrinous adhesions which may give rise to later intestinal obstruction.

Residual abscesses which may occur in both drained and undrained cases have been attributed both to drainage and to failure to drain. At Chirmont's Clinic in Zurich according to Ochsner a much higher proportion of residual abscesses was observed when drainage was used prophylactically probably because the drain acted as a foreign body thus increasing the peritoneal reaction and tending to produce pocketing. Vile expressed the opinion that no form of prophylaxis can prevent all rectovaginal abscesses. In his 26 patients fluid was removed by suction at the first operation in all 7 were drained to the pelvis 16 had drainage in the right lower quadrant and in the 3 other patients drainage was omitted.

*Clinical Picture and Diagnosis* The symptoms and signs of residual intra-abdominal abscesses vary somewhat according to the location of the abscess but do not differ greatly in character from the signs and symptoms of primary appendiceal abscess (p. 201) except that they appear as a rule after a period of satisfactory convalescence. They include pain nausea and vomiting fever rapid pulse and localized tenderness. The fever is frequently hectic in untreated cases.

Localization of internal abscesses in the absence of masses or localizing symptoms is not always easy. Roentgenologic examination has

been advised by numerous observers Beck Kouchy and Baker described a systematic plan of procedure After fluoroscopy has been carried out a scout film is taken with the patient on his back Other films are taken in the upright position on the left side and finally in the right lateral decubitus A sufficiently large abscess will be demonstrated as an area of opacity displacing loops of air filled bowel The authors advise that in difficult cases the cecum be distended by the cautious injection of a small amount of air into the colon through a rectal tube but this method has obvious dangers Failure of the patient to improve as would be expected after evacuation of a primary abscess should lead to the suspicion that a residual abscess has developed

*Therapy* Residual abscesses regardless of their location should be treated on general surgical principles which implies the release of pus plus adjuvant measures (p 298) Incision and drainage should not be delayed too long for the rupture of an abscess into the peritoneal cavity is a serious possibility (p 206) and is fraught with serious consequences Technical methods are discussed under the appropriate heading (p 258)

#### PELVIC ABSCESS

A pelvic abscess once it is suspected is the easiest of all varieties to diagnose but because of its location it is frequently overlooked until it is of considerable size The usual symptoms and signs of suppuration may be less pronounced than in other types of abscess There may be slight hypogastric distention but rigidity is frequently absent and the temperature may be normal Signs of toxicity are also less though whether because absorption is less from the pelvic peritoneum than from other portions of the cavity it is not possible to say

The most typical symptom of pelvic abscess is irritation of the anterior rectal wall with resulting tenesmus diarrhea and perineal distress In fact the mere appearance of diarrhea in either surgical cases or cases treated expectantly should suggest the diagnosis Mucus in the stools is also characteristic Rectal symptoms are not relieved by defecation If the abscess impinges on the bladder urinary symptoms are frequent Vale found distention of the small intestine often with extensive ballooning of the bowel in 9 of 26 cases in which colicky pain nausea vomiting increased and audible peristalsis and even plain roentgenogram suggested a diagnosis of obstruction

The diagnosis is made by repeated examinations of the rectum which should be carried out at least every 48 hours preferably by the same surgeon with the bowel and bladder empty Rectal irritation and

tenderness may be apparent before a definite mass can be made out because the purulent collection may gravitate to the pelvis immediately after rupture has occurred

In early stages an area of softening is felt which is painful on pressure. In late stages a large mass may bulge into the rectum. The anal sphincter is relaxed and edema of the genitals which is especially frequent in children may give evidence of pressure. In children the shallowness of the cul de sac sometimes permits abdominal demonstration of the mass. Proctoscopic examination reveals the rectal mucosa to be inflamed, edematous and succulent but it is not necessary in most cases.

One characteristic of pelvic abscess is an elevation fall and subsequent rise of temperature as the abscess forms is resolved and forms again.

#### ILEOCECAL ABSCESS

Ileocecal abscesses present the symptoms of primary appendiceal abscesses in this location that is they are likely to suggest gastroenteritis, dysentery or intestinal obstruction. They are also as likely to precipitate intestinal obstruction because of kinks, adhesions and edema as are primary appendiceal abscesses. The rupture of such an abscess into the peritoneal cavity if operation is delayed too long has particularly serious consequences.

#### LEFT SIDED ABSCESS

Left sided abscess may develop as the result of a situs inversus or of a long appendix extending to the left or because a spreading peritoneal process localizes on the left or because a cul de sac abscess rises out of the pelvis on the left side. The usual left sided abscess is bounded below and laterally by Poupart's ligament and above and medially by intestinal loops which must be separated with great care when operation is undertaken.

There were only 9 left sided abscesses in 700 cases of acute appendicitis observed by Nather and Ochsner over a 4 year period at the University of Zurich Clinic. Four of these occurred in children under 13 years of age and Royster states that two thirds of his personal cases occurred in children under 12 years of age. Left sided abscesses in children are usually on a somewhat higher level than in adults and are chiefly found on a line connecting the anterior superior spines of the ilia.

## SUBPHRENIC SPACE INFECTION

Subphrenic (subdiaphragmatic) space infection is one of the most serious complications of acute appendicitis. The incidence is variously reported. It varied from 6.6 to 7.3 per cent in the residual abscesses reported in the individual series which made up the more than 10,000 cases of acute appendicitis studied by Ochsner, Gage and Garside. Ochsner and DeBakey found 135 subphrenic abscesses in 14,969 collected cases of acute appendicitis, an incidence of 0.9 per cent which seems nearer the average. The Charity Hospital figures support the point of view that this complication is not very frequent. Subphrenic space infection was responsible for or was found in only 4 of 320 surgical deaths from acute appendicitis at the New Orleans Charity Hospital over a 15½ year period and was proved or suspected on reasonable grounds in only 33 other cases among the 6,121 surviving patients treated surgically over this period. It is true that many subphrenic space infections subside without abscess formation but the question always arises in such instances whether the diagnosis was correct. Many times the complication is suspected when it is not present and is overlooked when it is as the following case histories indicate.

*Case 19.* A colored man 25 years of age had had attacks of pain in the midclavicular line below the right costal margin for 3 months. The attacks at first occurred once or twice weekly and lasted only 30 or 40 minutes. Recently they had been more frequent and had lasted for several hours. The pain was sharp and sticking in character, was worse at night, was not aggravated by movement and for the past several days had radiated into the epigastric area. After the acute attacks subsided there was always tenderness in this region. The patient was often nauseated but had vomited only once shortly after the first attack of pain. The temperature was 98.6° F., the pulse rate 82 and the respiratory rate 22. Physical examination was negative except for slight tenderness over McBurney's point. The white blood cell count was 25,750 per cu. mm. with 82 per cent polymorphonuclear leukocytes.

The patient did not seem acutely ill and a diagnosis of chronic appendicitis was made. Three days later appendectomy under spinal anesthesia through a McBurney incision revealed an acutely inflamed appendix.

Following operation the patient had daily temperature elevations from 103° to 105° F. with a pulse rate averaging 130. The respiratory rate ranged from 20 to 28. Repeated examinations revealed no explanation for the temperature elevation. The wound was clean and well healed when the sutures were removed on the seventh postoperative day. Roentgenologic examination on the twelfth day revealed an elevation of the right leaf of the diaphragm. Two days

tenderness may be apparent before a definite mass can be made out because the purulent collection may gravitate to the pelvis immediately after rupture has occurred

In early stages an area of softening is felt which is painful on pressure In late stages a large mass may bulge into the rectum The anal sphincter is relaxed and edema of the genitals which is especially frequent in children may give evidence of pressure In children the shallowness of the cul de sac sometimes permits abdominal demonstration of the mass Proctoscopic examination reveals the rectal mucosa to be inflamed edematous and succulent but it is not necessary in most cases

One characteristic of pelvic abscess is an elevation fall and subsequent rise of temperature as the abscess forms is resolved and forms again

#### ILIOCECAL ABSCESS

Ileocecal abscesses present the symptoms of primary appendiceal abscesses in this location that is they are likely to suggest gastroenteritis dysentery or intestinal obstruction They are also as likely to precipitate intestinal obstruction because of kinks adhesions and edema as are primary appendiceal abscesses The rupture of such an abscess into the peritoneal cavity if operation is delayed too long has particularly serious consequences

#### LEFT SIDED ABSCESS

Left sided abscess may develop as the result of a situs inversus or of a long appendix extending to the left or because a spreading peritoneal process localizes on the left or because a cul de sac abscess rises out of the pelvis on the left side The usual left sided abscess is bounded below and laterally by Poupart's ligament and above and medially by intestinal loops which must be separated with great care when operation is undertaken

There were only 11 left sided abscesses in 700 cases of acute appendicitis observed by Nather and Ochsner over a 1 year period at the University of Zurich Clinic Four of these occurred in children under 13 years of age and Royster states that two thirds of his personal cases occurred in children under 12 years of age Left sided abscesses in children are usually on a somewhat higher level than in adults and are chiefly found on a line connecting the anterior superior spines of the ilia

derness most marked on the right side moderate generalized rigidity and a possible right sided mass The white blood cell count was 13 250 per cu mm with 90 per cent polymorphonuclear leukocytes

The admission diagnosis of pelvic inflammatory disease was soon changed to appendicitis with rupture and the patient was treated conservatively for 10 days by standard measures On the eleventh day incision and drainage of a small appendiceal abscess was carried out under local analgesia The patient died 3 days later

Postmortem examination revealed a ruptured retrocecal appendix generalized peritonitis a pelvic abscess abscesses of the subhepatic and suprahepatic spaces right sided empyema and bilateral hydrothorax

*Anatomy* The anatomy of the subphrenic space has been clarified by the studies of several observers whose combined investigations supply the following summarized data

The subphrenic space as its name implies is located beneath the arch of the diaphragm It is covered by the thoracic cage and is therefore not readily available for either examination or treatment It is limited above by the diaphragm and below by the transverse colon and its mesentery

For all practical purposes the subphrenic space (fig 51) is divided into

- 1 The intraperitoneal and extraperitoneal areas

- 2 The suprahepatic and infrahepatic areas which are formed by the protrusion of the liver into the intraperitoneal area

- 3 Three suprahepatic intraperitoneal areas composed of (a) the left suprahepatic area located above the liver and to the left of the coronary ligament (b) the right anterior suprahepatic area located just above the liver to the right of the coronary ligament and anterior to the right lateral ligament (c) the right posterior suprahepatic area located just above the liver and behind the right lateral ligament

- 4 Three subhepatic (infrahepatic) intraperitoneal areas composed of (a) the right infrahepatic area located inferior to the liver and to the right of the hepatic round ligament and the ligament of the ductus venosus (b) the left anterior infrahepatic area located inferior to the liver to the left of the hepatic round ligament and the ligament of the ductus venosus and anterior to the stomach (c) the left posterior infrahepatic area located inferior to the liver to the left of the round ligament and the ligament of the ductus venosus and posterior to the gastrohepatic omentum

- 5 Three extraperitoneal areas composed of (a) the anterior extraperitoneal area located between the anterior external surface of the

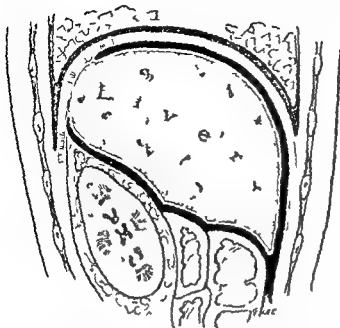


Fig. 51 Diagrammatic showing of subphrenic area in which infection following acute appendicitis is most likely to occur

Later the patient became jaundiced and began to complain of dyspnea and epigastric pain. Abdominal distention developed rapidly. Thoracentesis revealed bloody fluid and a tentative diagnosis was made of liver abscess with rupture into the pleural cavity.

The patient did not respond to transfusion, infusion, intestinal decompression and similar measures. Coffee grounds material was regurgitated in large quantities. When the jaundice disappeared the diagnosis was changed to subphrenic abscess with rupture into the pleural and peritoneal cavities. Death occurred on the fifty-fifth postoperative day.

Postmortem examination revealed multiple liver abscesses with rupture into the pleural cavity and generalized peritonitis. The portal vein was clean throughout its course. The original diagnosis of liver abscess which was later changed to subphrenic abscess was proved by postmortem examination to be correct.

**Case 20** A colored female 27 years of age had a sudden attack of cramping pain 3 days before her admission to the hospital while eating her dinner. Vomiting followed. She came to the hospital a few hours later but by that time was considerably better and she was therefore not admitted. Twelve hours later she returned complaining of precordial pain and dyspnea. She had taken salts immediately after the onset of the pain. The temperature was 101° F, the pulse rate 104 and the respiratory rate 24. The blood pressure was 107/70. The patient was now acutely ill. Examination of the chest was negative except for very shallow breathing. Abdominal examination revealed generalized ten-

- 2 As a result of a residual infection after spreading peritonitis
- 3 As a result of multiple liver abscesses which result in turn from pyelphlebitis
- 4 As a result of retroperitoneal lymphangitis If an appendix adherent to the posterior parietal peritoneum is incompletely removed and infected necrotic tissue is left *in situ* or if a part of the peritoneal surface is torn away when an infected appendix is removed a raw infected area is left through which organisms may reach the retro cellular spaces and lymphatics and thence the subdiaphragmatic space (Hochberg)

Although the etiology of subphrenic space infections is not clear in all cases appendicitis is probably the most frequent cause These infections occasionally develop after simple acute or acute suppurative appendicitis especially if the appendix is retrocecal but most often they follow advanced disease associated with spreading peritonitis or with abscess

Appendicitis was responsible for 35 of the 111 subphrenic space infections reported by Faxon in 26 per cent of which the etiology was not clear and for 26 of the 111 cases reported by Hochberg in 15.3 per cent of which the etiology was not clear In 3 608 cases collected from the literature by Ochsner and DeBakey including 75 personally observed cases appendicitis was responsible for 30.9 per cent

Although infection may occur in any subdivision of the subphrenic space the right posterior superior space (fig 52) is most often affected It was involved in more than a third of the cases collected by Ochsner and DeBakey in which the exact location was noted and in well over half of their personal cases In 111 cases studied by Faxon 87 per cent occurred on the right side and 38 per cent in the right posterior superior space In 90 surgical cases studied by Hochberg however 77.7 per cent of the cases occurred on the right side but only 20 per cent in the right posterior superior space The left inferior anterior and the right anterior superior spaces are probably involved next in order of frequency the proportion varying somewhat in the reported series There is fairly general agreement that extraperitoneal involvement is least frequent

The anatomic reason for the frequency with which the right posterior superior space is involved in cases of appendiceal etiology is the accessibility of this space to an inflammatory exudate which may extend upward from the iliac fossa along the paracolic gutter Hochberg emphasized that the source of the infection and the time at which the patient is seen will finally determine the location of the infection He



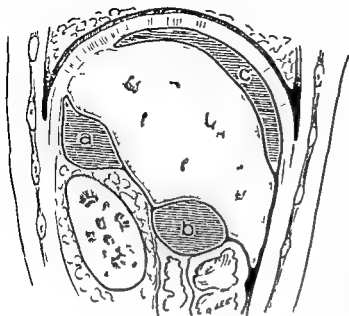


Fig 52 Diagrammatic showing of location of most frequent subphrenic space infections a, right posterior space b subhepatic space c right anterior space

parietal peritoneum and the internal surface of the superior anterior abdominal wall below the diaphragm (b) the right posterior extraperitoneal area located between the posterior external surface of the parietal peritoneum and the superior posterior abdominal wall below the diaphragm and to the right of the inferior leaf of the coronary ligament this area including the bare area of the liver (c) the left posterior extraperitoneal area located between the posterior external surface of the parietal peritoneum and the superior posterior abdominal wall inferior to the diaphragm and to the left of the inferior leaf of the coronary ligament

*Etiology and Pathology* Exactly how subphrenic space infection occurs is not always clear but the following routes have been suggested and in some instances proved

1 As a result of ascending infection along the paracolic gutter to the right kidney pouch and the subphrenic space which is probably the most frequent route Extension by this channel is favored by the presence in the upper abdomen of negative pressure fluctuating with respiration and sucking infection upward (Overholt and Donchess) as well as by the ventral curvature of the spine which when the patient lies supine presents as a transverse ridge so that pus gravitates distally into the pelvis and proximally into the subphrenic region The tendency may be aggravated by the use of the Trendelenburg position

may extend to the lumbar region near the vertebral column to the upper abdomen or to the suprahepatic region all areas which are supplied by the phrenic nerve. When the right posterior superior space is involved the patients usually complain of pain or soreness in the right flank (Ochsner). Nausea and vomiting may be present. Later the temperature is of the swinging type and there may be chills and drenching sweats. Hiccoughs and a hacking cough are fairly usual.

The patient looks acutely ill. The face may be pale or flushed but one always receives an impression of extreme toxicity. The expression is apprehensive. Inspection of the chest may show the involved side moving only slightly if at all. If the abscess is near the surface the interspaces are widened and bulging. If it is deeply placed they may be retracted. The costal arch over the involved area is tender. If the abscess is located in the right posterior suprahepatic space there is tenderness along the right twelfth rib posteriorly and a definite localized point of tenderness over the tip of this rib is an early and reliable diagnostic sign (Ochsner and DeBakey). If the abscess is in the anterior infrahepatic space the tenderness will be located along the tenth rib anteriorly (Hochberg). There may be edema over the affected area (Overholt and Donchess).

Percussion which may be painful reveals impairment over the lower part of the chest and distant or suppressed breath sounds in this area. A friction rub may be heard in the same area. Sometimes there is evidence of fluid. If the abscess is on the left side and is of considerable size the heart is pushed upward or tilted to the right but is not displaced in the ordinary sense of the term. If the abscess is on the left side there is occasionally a peculiar clicking quality to the second sound at the apex (Hochberg). Although any or all of these findings may be observed Ochsner warned against placing too much reliance on their presence or absence since clear cut pulmonary signs and symptoms can be expected only in long standing cases.

Abdominal examination shows an absence of movement on the affected side, spasticity of the rectus muscle and if the abscess is suprahepatic downward displacement of the liver. Hochberg outlined four distinct zones of sound on percussion: these being from above downward a zone of normal resonance or hyperresonance in the lung, a zone of pleural effusion or compression of the lung with dullness, a zone of resonance or tympany due to gas and a zone of dullness due to the abscess itself.

Examination of the lumbar region may reveal a tender usually tense and sometimes fluctuant mass extending out from below the

also pointed out that following appendicitis an infection may first be located in the right subhepatic space and later in the right posterior subhepatic space the manifestations of the secondary extension becoming pronounced after the primary subphrenic infection has begun to subside. There was evidence to corroborate this chain of events in 3 of the 90 surgical cases in his series.

Subphrenic abscesses are less usual on the left side after appendicitis than after other lesions but can occur as Carter demonstrated by the report of 2 cases. He suggested that the adage, Pus somewhere pus nowhere, pus under the diaphragm be modified to Pus somewhere pus nowhere, pus not under the right half of the diaphragm, pus under the left half of the diaphragm.

Bacteriologic studies have revealed the causative organisms in subphrenic abscess to be chiefly *Escherichia coli*, *Streptococcus* and *Staphylococcus*. Multiple organisms are present in from a quarter to a third of all cases.

**Clinical Picture and Diagnosis.** There is no classic picture of subphrenic abscess. The etiologic factors, the location of the abscess, the duration of the illness, and even the age of the patient, all are likely to alter the manifestations. The age range in Ochsner and DeBakey's collected series was 14 months to 76 years, and it is obvious, aside from any other consideration, that the factor of age, when the range is so wide, is likely to introduce confusion. Diagnosis is simpler in postoperative cases than in primary subphrenic abscess. The disease is more likely to be suspected, and suspicion is the first step to diagnosis.

Subphrenic space infection is most frequent in cases in which the postoperative course, because of the advanced degree of the original disease, has been stormy, and the initial symptoms of the subphrenic infection are sometimes overshadowed by the symptoms of the primary infection. Less often the primary disease is mild or convalescence has proceeded satisfactorily for a period of days or even for 2 or 3 weeks before symptoms develop.

Although the onset of subphrenic abscess is insidious and there are no pathognomonic manifestations, the condition should be suspected in any patient who has had an intra-abdominal infection and whose clinical course indicates that it has not been controlled. The first symptoms are malaise, irritability, fatigue, anorexia, and a general lack of well-being. In the beginning the symptoms are not localized and could scarcely be expected to be, because of the tendency of the infection to overlap adjacent areas instead of being limited to a single area. The first localizing symptom is likely to be a dull ache in the thorax, which



Fig 54 Subdiaphragmatic space infection. Note downward displacement of hepatic flexure of colon.



Fig 55 Roentgenogram of thorax and upper abdomen in case of subhepatic and suprahepatic space infection. Note displacement of diaphragm, fluid levels and gas shadow.

chest in all patients with acute appendicitis for comparison with later films if subphrenic abscess should be suspected. The demonstration of gas and of a fluid level is important but these findings occur in only a small proportion of all cases variously estimated at from 15 to 35 per cent. The taking of serial roentgenograms from all aspects for comparative purposes is a very valuable diagnostic procedure.

Examination of the blood usually reveals anemia which may be marked, a leukocytosis above 20,000 per cu mm and a polymorphonuclear percentage of 85 or higher. There is however nothing distinctive about these findings.

The differential diagnosis lies chiefly between pleurisy in early cases and empyema in later cases. As a matter of fact an irritative pleurisy usually develops in most untreated cases probably as the result of the passage of toxins through the diaphragmatic lymphatics. The apparent development of pleurisy in a patient recovering from appendicitis therefore demands the exclusion of subphrenic abscess before the diagnosis of pleurisy is made. DaCosta (cited by Hochberg) considered that the chief differential point between empyema and subphrenic abscess is that in the former the upper limit of fluid is concave whereas in subphrenic abscess it is convex.



Fig 53A Roentgenogram of chest and upper abdomen in case of right subdiaphragmatic space infection. Note elevation of diaphragm which is frequently of earlier diagnostic value than the physical examination. Note fluid level and gas shadow.



Fig 53B Lateral roentgenogram of right subdiaphragmatic space infection shown in 53A.

costal margin posteriorly. The mass which may be so large as to reach around to the navel line is dull to percussion unless it contains gas, then it is hyperresonant. Pressure will cause local pain which may be referred to the shoulder, epigastrium, or anterior infrahepatic region on the affected side. Hochberg, from whom the preceding data have been quoted, stated that the onset of subphrenic abscess occurred with symptoms referable to the lumbar region in 25 per cent of his 111 cases.

Roentgenologic examination (figs 53-5) is the most valuable diagnostic method available, and fluoroscopy should never be omitted. When the patient is examined in the upright position, the diaphragm will be seen to move very little during respiration and to be elevated and flattened at its dome. Some observers believe that no condition other than subphrenic abscess completely inhibits the movement of the diaphragm, and therefore regard inhibition of diaphragmatic movement as pathognomonic. Beck and his associates stated that if fluid levels are present in the cavity, a right lateral film with the patient upright will help to determine whether the abscess is anterior or posterior. Walker advises routine roentgenologic examination of the

abscess may follow the breakdown of infected mediastinal glands or may result from direct extension from a subdiaphragmatic abscess or pneumonitis. In other words these and other complications show that subphrenic space infection is a condition in which for anatomic reasons one complication is likely to lead to another complication and in which the patient's risk is cumulative.

**Therapy** The prophylaxis of subphrenic space infection of appendiceal origin consists in prompt operation on the inflamed appendix. This is frequently a matter beyond the surgeon's control. His direct responsibility therefore usually begins at operation at which by careless technique and other errors many infections are initiated. Prophylaxis is extremely important. The involved appendiceal area should be thoroughly willed off at all operations. All maneuvers should be gentle. Every attempt should be made to remove all the infected tissue and to leave no raw areas particularly on the posterolateral parietal peritoneum. Drainage should perhaps be instituted in some cases of generalized and localized peritoneal infection though the statement is made with some hesitancy in view of the excellent results achieved by many surgeons without drainage and in view of the favorable effects of modern chemotherapy and antibiotic therapy. Postoperative postural drainage is an excellent means of prophylaxis and for purely mechanical reasons there should be a decreased incidence of subphrenic space infections when this method is employed. The preservation of the proper fluid balance and other adjuvant measures are also prophylactic (p. 298).

Similar supportive measures should be carried out in cases in which subphrenic space infection is suspected but in which there is no warrant for exploration. Hochberg advocated the Fowler position, the use of dry heat locally and the strapping of the lower part of the chest on the affected side to inhibit the suction effect of the diaphragm. Chemotherapy and antibiotic therapy should be routine. If the patient does not improve under these measures and if there is reason to suspect that pus is present surgical exploration should be resorted to promptly, the operation of choice being an extraperitoneal procedure (p. 287).

**Prognosis** An infection of the subphrenic space may subside spontaneously and the same outcome is possible though less frequent in definite abscess formation. In such cases as has been mentioned one can not always be positive that infection has really been present. The outcome of an untreated case of true subphrenic infection is not usually good however and a high incidence of complications can be expected even if recovery ensues. The virulence of the infecting organisms, the location of the infection, the patient's resistance, the duration of the

In the usual case there is no justification for aspiration or exploratory puncture. The risk of contaminating the pleural or peritoneal cavity is serious and the mere failure to find pus does not exclude the diagnosis. Surgical exploration by the extraperitoneal route (p. 287) is attended with far less risk. Both pneumoperitoneum and the injection of thorotrast into the abscess cavity have been suggested as diagnostic aids but are not in general use.

**Complications.** Cases of subphrenic abscess in which treatment has been delayed are likely to show a high incidence of complications, the number being in proportion to the delay in diagnosis and treatment. Systemic complications including sepsis and cachexia do not differ from those which would be expected in any infectious process of similar severity. Abdominal complications include rupture of the abscess into the peritoneal cavity with subsequent peritonitis or rupture into the stomach, colon or other adjacent viscera. Rupture through the skin surface is also a possibility.

Opinions differ as to whether pleurisy or pleural effusion is the most frequent intrathoracic complication of subphrenic abscess. In Ochsner and DeBakey's collected series pleurisy was most frequent and effusion was not observed in any of their personally treated cases though other authorities believe that it is likely to be present in most cases. Empyema may be a complication of serous pleural effusion or may be secondary to lung abscess, pneumonia or sepsis. It may also result from direct infection of the pleural space from rupture of the subphrenic abscess into the pleura or from entrance of organisms into the pleura by way of the supra-diaphragmatic lymphatics.

Lung abscess is usually the result of lymphatic extension of the process into the lung but may be of postpneumonic or embolic origin. Pneumonia or pneumonitis is either a superimposed infection or an extension of the subdiaphragmatic process. Bronchopleural fistula is usually secondary to empyema, pneumonia or pulmonary abscess.

Perforation of the diaphragm by a subphrenic abscess will result in empyema or pneumonitis and bronchial fistula depending upon whether or not a free pleural space exists. Empyema is likely to result if the parietal diaphragmatic pleura is not adherent to the visceral pleura at the base of the lung. If these pleural surfaces are adherent rupture occurs into the lung with the development of a basilar pneumonitis and bronchial fistula (Ochsner and DeBakey).

Pericarditis is usually the result of a lymphatic extension of the infection from below the diaphragm especially on the left but may be secondary to empyema, pneumonitis or lung abscess. Mediastinal

abscesses is slightly greater on the right side than on the left Pyemia is also a possible outcome

It has been suggested that pylephlebitis may extend directly from the original area of suppuration by way of the lymphatics to the portal vein or that the process may reach the liver by way of the loose retrocecal areolar tissue Both routes are possibilities but extension from the mesentery probably occurs in the great majority of all cases

Phlebitis of the portal vein occurs most often in older persons and in advanced and neglected forms of acute appendicitis particularly in association with gangrene abscess and peritonitis It is always a possibility when the mesenteric omentum is friable and thickened and the retroperitoneal tissues are edematous Snyder and his associates observed in some of their cases of gangrenous appendicitis that when the mesentery was cut there was little or no bleeding which suggested that occlusion of the veins had already occurred Further investigations however showed that the veins were plugged with septic material in 10 per cent of the general run of cases and these observers therefore concluded that there was no way of determining which cases would prove serious and which would not In other words thrombi that remain uninfected seldom give rise to further trouble

*Clinical Picture and Diagnosis* Pylephlebitis does not usually manifest itself for at least 72 hours after operation and may appear as late as 4 or 5 weeks afterward Less often it occurs earlier as in the case reported by Keyes in which it developed within 24 hours after the onset Lewis and Firor reported a similar case

Chills are the most typical symptom of pylephlebitis and their occurrence after operation is highly significant though their appearance either before or after does not necessarily presage its development Colp who studied his cases from this aspect found 2 deaths from pylephlebitis in 181 cases in which a single chill had occurred and 4 deaths in 11 cases of multiple chills Two of 3 patients who had chills after operation but who had none before also died of this complication

Chills occurred in 4 of the 6 fatal cases of pylephlebitis (2 surgical 4 nonsurgical) observed at the New Orleans Charity Hospital They also occurred however in 17 other fatal nonsurgical cases and in 259 other surgical cases in which this complication did not develop there were 22 deaths in the surgical group 8.5 per cent In view of the relatively high proportion of deaths in cases in which chills are present and the relatively small incidence of pylephlebitis even when only autopsied cases are considered it seems reasonable to attribute most



illness and the adequacy and skill of surgical intervention all help to determine not only the outcome from the standpoint of life or death but also the incidence and seriousness of the complications

It is clear from reported results that surgery must be employed in the great majority of cases in which suppuration has occurred. At that the mortality is high. In Ochsner and DeBakey's collected series the mortality without surgery (which was most often omitted because the diagnosis was not made) was 89.8 per cent in 1,096 cases as compared with 32.8 per cent in 1,942 cases treated surgically. In the 111 cases which Hochberg observed the total mortality was 21.6 per cent, 17.6 per cent for the 90 cases in which operation was performed and 38.4 per cent for the 21 in which it was omitted.

### PHLEPHLEBITIS AND LIVER ABSCESS

Pylephlebitis (suppurative thrombosis of the portal vein) is a highly fatal but fortunately infrequent complication of acute appendicitis. The incidence is variously estimated. Fitz observed it in 11 of his 257 postmortem cases. Colp in 9 of 2,841 cases and in 6 per cent of his fatal cases. Armstrong in 5 per cent of 546 postmortem cases. Koster in 6 of 4,561 cases and Snyder and his associates in 27 of 8,969 cases.

Pylephlebitis was responsible for the fatal outcome in 2 of 320 surgical deaths from acute appendicitis at the New Orleans Charity Hospital and in 4 of 158 nonsurgical deaths, in 2 of which it was associated with liver abscesses. The incidence is small in all reported series but the relatively high incidence reported in the Charity Hospital nonsurgical autopsied cases (4 of 67 cases) makes it evident that autopsy figures are the only ones to be relied upon and suggests that the incidence is really higher than statistics based on clinical observation would suggest.

**Pathologic Process.** The initial process of pylephlebitis is in the small veins of the appendiceal mesentery.\* Hawkes took the position that thrombophlebitis of the appendiceal vein so alters the diagnostic, pathologic, prognostic and therapeutic features of appendicitis that a special category should be made for this type of case. Extension (fig. 20 p. 33) occurs successively into the ilioocolic vein, the superior mesenteric vein and finally the portal vein from which in many instances it extends into the liver where it gives rise to multiple abscesses or occasionally to a single abscess. The incidence of hepatic

cases and called attention to the enlargement of the spleen often noted at autopsy though this is not an important clinical finding

Examination of the blood shows a high leukocytosis and serum bilirubin and similar tests may demonstrate jaundice before it is evident clinically Liver function tests are occasionally useful Blood cultures are usually negative and would not be expected to be positive until abscesses in the liver had caused an infective thrombophlebitis of the branches of the hepatic veins Then bacteria appear in the general circulation for the liver has lost its bactericidal function and the lungs do not serve as an effective filter (Thalheimer)

Koster recommended the intravenous injection of thorium dioxide (in 25 cc doses on 2 successive days) in the diagnosis of pylephlebitis having used it himself with good results and without harm for 17 years The opaque substance is picked up by the reticulo endothelial system In the normal subject the liver shadow on the roentgenogram is of uniform density If a single liver abscess is present the cavity is outlined as an area of lesser density and if multiple abscesses are present the shadow is mottled Yater also reported good results from the same diagnostic method but preferred to inject the thorotrast (75 cc) in a single dose and to take the exposures 2 hours or more later

Bassler reported 3 cases of pylephlebitis in which diagnosis was made by the use of a barium meal The meal was markedly slowed in its passage from the jejunum to the middle portion of the transverse colon this being the area supplied by the superior mesenteric vein The method is obviously not adapted to seriously ill patients who are not likely to be taking fluids or food by mouth Roentgenologic examination however will often show a limitation of the diaphragm on the right side

*Treatment* The treatment of pylephlebitis and liver abscess following acute appendicitis was until the introduction of modern chemotherapeutics and antibiotics a practically hopeless task Barlow in 1940 called attention to a recovery which had occurred in the Naval Hospital Plymouth in 1919 following the use of intravenous eusol and which had been authenticated by Sir Humphrey Rolleston but the mortality was usually regarded as 100 per cent Drainage of a solitary liver abscess is possible but surgery is clearly of no benefit in multiple abscesses The extreme seriousness of pylephlebitis led to numerous radical suggestions for its management including such measures as prophylactic ligation or resection of the ileocolic vein and ligation of the ileocolic artery the superior mesenteric veins and even the portal vein

The introduction of the sulfonamide drugs and penicillin has altered

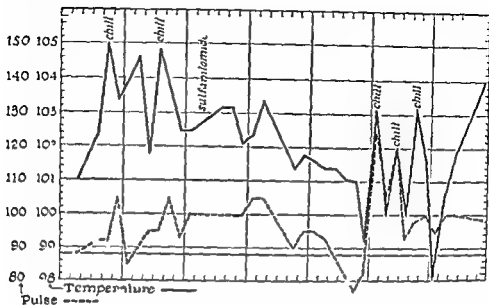


Fig 56 Typical temperature chart in pykphlebitis following acute appendicitis

of the high mortality associated with chills to delay in diagnosis and treatment because of the diagnostic confusion which they introduce (p 119)

Regardless of whether or not chills presage the development of pykphlebitis there is no doubt that they frequently precede this complication and a special effort should be made to diagnose and treat without loss of time patients who are having chills and in whom the diagnosis of acute appendicitis is suspected. Chills are not usual in simple acute disease and their presence may indicate an advancing pathologic process especially gangrene.

The patient with pykphlebitis is likely to have a high spiking temperature (fig 56) and exhausting sweats. He is anorectic and extremely toxic. Jaundice may or may not be present and may be slight or profound. There may be severe irregular pain in the right upper quadrant. Distention occurs late and ascites is a terminal phenomenon and not likely to be present until portal obstruction is complete.

Physical examination shows a seriously ill patient. Jaundice may be absent, present only in the sclera, or generalized. The liver is enlarged and tender and compression of the right lower lung often results from upward displacement of the diaphragm, but the chest findings are negative. The abdomen is surprisingly free of positive findings in many cases. Roster was able to elicit tenderness over the portal vein in some

## (X)

### *The Complications of Acute Appendicitis*

#### II OTHER COMPLICATIONS

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##### WOUND INFECTION

In the uncomplicated case of acute appendicitis wound infection can usually be charged to carelessness in surgical technique chiefly in the form of inadequate protection of the wound edges during operation. Occasionally even in the absence of technical errors it develops in obese patients who are peculiarly prone to fat necrosis. Most often however wound infection is the result of contamination of the edges of the incision during the removal of a gangrenous or perforated appendix or of contamination of the incision by drainage of infected material from a ruptured appendix or an appendiceal abscess. Even in the hands of careful surgeons it is likely to occur in a large proportion of such cases. A frequently unsuspected cause is the dressing of wounds by a technique which does not include masking. As a matter of fact it is surprising that more infections are not thus caused for masking is not part of the usual technique of wound dressing. The infection may involve one or more stitches or the whole length of the incision and may be superficial or deep.

Wound infection is manifested clinically by local pain, fever and leukocytosis. Examination shows the usual signs of local inflammation that is heat, redness, tenderness and sometimes actual suppuration. Deep abscesses in the preperitoneal tissues beneath the fascia and muscle may be insidious in onset and may cause considerable tissue destruction before they are detected. Wound infection which has given rise to no definite local signs will often be found to be the cause of continuous unexplained fever beginning a few days after operation. The wound should be promptly investigated in such cases.

*Treatment* If the infection is localized around a single stitch or several stitches removal of the suture material is usually all that is needed. If a definite stitch abscess has formed drainage can be achieved by the insertion into the abscess of a pair of forceps which are then

the picture in pylophlebitis and liver abscess following acute appendicitis. The first cases successfully treated by sulfanilamide were reported by Ottenberg and Berck in 1938 and other survivals have since been recorded by Gamm, Wilensky, Murray and Donelson and others. The outlook was still further improved by the introduction of penicillin and of streptomycin. Pulaski reported a case in which recovery with uncomplicated wound healing followed the administration of 25 gm of streptomycin in divided doses daily for 14 days although when therapy was begun the patient was having recurring chills with temperature elevations to 106° F, was jaundiced and had an enlarged and tender liver. Wishart and Peterson reported an equally dramatic recovery.

The present therapeutic regimen is to attempt no surgery in pylophlebitis and liver abscess but to treat the patient by the usual adjuvant measures (p. 295) and to employ either streptomycin or large doses of penicillin combined with sulfadiazine according to the indications of the special case. This mode of treatment has greatly improved the prognosis but pylophlebitis and liver abscess are still highly fatal complications of acute appendicitis.

True *Clostridium welchii* infection is not usual after appendectomy whereas if gas forming organisms played as large a part in acute appendicitis as they were once supposed to play it would seem that it should be quite frequent. Of the 52 cases of wound infection due to this cause collected by Miller in 1932 only 10 followed appendectomy. No review of the literature from this standpoint has been attempted but a casual survey shows 2 cases reported since that date by Johnson, 6 by Eliason, Erb and Gilbert, and 1 each by Gordon and by Breslin. I have personally observed a case following appendectomy performed without inversion of the stump though it is doubtful that the technique had anything to do with the development of the infection. In the majority of reported cases in which pathologic data were stated the infection followed gangrene of the appendix in which conditions apparently are optimum for the growth of the organism. The infection originates within the intestinal tract in contrast to gas bacillus infection following trauma in which the origin is extrinsic.

In the typical case of *Cl. welchii* infection following appendectomy the patient has a sudden rise in temperature and pulse the second or third postoperative day and presents symptoms of overwhelming toxemia. There is severe pain at the site of the wound and examination reveals a coppery discoloration of the tissues. Exposed muscle is dark and lifeless and does not contract on stimulation. The discharge is dark reddish brown and is likely to have a foul mousy odor. It may or may not contain gas bubbles. Frequent observation is necessary for the infection progresses very rapidly and startling changes may be apparent within an hour.

The accepted treatment of gas gangrene is free drainage by multiple incisions with excision of affected tissues to whatever degree is practical. Not very much tissue can be excised from the abdominal wall. These surgical measures are supplemented by transfusion and the use of polyvalent serum and of penicillin. The place of serotherapy in gas bacillus infection remains to be clarified as does the use of penicillin. The military experience in World War II provided no conclusive answers but in a condition of such serious potentialities the use of both measures seems justified in the absence of measures of proved effectiveness. For the same reason the use of X ray therapy as advised by Kelly and Dowell seems justified. The prognosis is less grave when only the wound is involved but is poor when the peritoneal cavity is involved.

The spread of infection in some of the reported cases is striking. Gordon's patient had an appendiceal abscess which was treated conservatively with clinical improvement for 2 days. On the third day he

gently opened. If the entire wound is involved and if such simple measures as hot moist compresses do not bring about resolution all the sutures should be removed and the wound should be laid open. Promiscuous probing does more harm than good.

**Chemotherapy.** Soon after the introduction of the sulfonamide drugs the policy of using them locally in the treatment of infected wounds became fairly general and some surgeons recommended that they be used routinely as a prophylactic measure in clean wounds. There was never universal agreement about the wisdom of these policies even among those who followed them. It was fully realized that the presence of pus inhibits the action of the sulfonamides and that undesirable toxic effects may occur no matter by what route the drugs are employed. It was also realized that any powder or other preparation introduced into a wound is a foreign body and that no matter how desirable its chemotherapeutic effects may be it also has all the undesirable effects of a foreign body.

The experience of World War II justifies the abandonment of local chemotherapy in practically all wounds. Experimental and clinical evidence well summarized by Lyons is to the effect that this method is of no value in preventing local infection and of no value in its management if it has become established. An infection which does not respond to simple measures such as the removal of sutures, the puncture of localized pus collections and hot compresses usually demands a surgical procedure. In such cases the systemic use of some chemotherapeutic (or antibiotic) agent is of great value in keeping an invasive infection within bounds and this method should be resorted to without delay in any infection serious enough to warrant it. It should not be employed in trivial infections which can readily be controlled by simpler means. Administration is by the same routine as in any other type of infection (p. 32).

#### CLOSTRIDIUM WELCHII INFECTION

Wilkie first called attention to the peculiar susceptibility of obese subjects to wound infection, especially anaerobic infection which takes the form of cellulitis. In cases which have been drained and in which this type of infection develops, drainage tends to cease about the fourth or fifth day and foul pus is evacuated at the side of—not through—the drainage tube. Investigation reveals necrosis of fat, aponeurosis and muscle. An extensive process may be sufficient to turn the scales against the patient.

The gangrenous skin is firmly adherent to the purple zone and there is very little undermining of the normal skin. As the process advances the gangrenous skin liquefies on its inner margin leaving exposed a base of granulation tissue which gradually enlarges. Patches of regenerating epithelium are observed in the areas in which the destruction of the dermis is not complete. In some of the reported cases the whole surface of the abdominal wall was involved in others the lesion spread downward to the thighs and in one instance it extended to the back.

Constitutional symptoms are remarkably mild in view of the extent of the lesion and fever is not very high. On the other hand the pain caused by the slightest manipulation of the involved area as well as the persistent extension of an unchecked process eventually wears the patient out. Death occurred in 8 of the 41 cases collected by Liedberg. Diagnosis which is by laboratory methods is not usually made until the process has become extensive.

*Therapy* The treatment of progressive postoperative gangrene of the abdominal wall has until recently been extremely unsatisfactory. All forms of conservative treatment promptly proved useless as did local excision of the lesion. Melencys practice as soon as the diagnosis was established was to excise the entire affected area including the outer zone of redness with the knife or the cautery. Plastic surgery was employed to correct the defect after a sufficient period of time had elapsed to make it clear that the infection had been controlled. Transfusions and other supportive measures were part of the therapeutic regimen.

Melency also advised the application of zinc peroxide. The drug is sterilized in quantities of not more than 5 to 15 gm. at 140° C. dry heat for 4 hours. The dry powder is suspended in approximately equal quantities of sterile distilled water so that it has the consistency of cream. It is applied with an aseptic syringe to every part of the wound surface which is then covered with a double layer of fine mesh gauze soaked in the suspension of zinc peroxide. This layer is covered with gauze compresses or with cotton wet with sterile distilled water and the whole dressing is sealed with several layers of vaseline gauze to prevent evaporation. The wound is dressed every 24 hours the exudate and old zinc peroxide being removed with saline solution before a fresh application is made. The treatment is continued until the whole area is covered by healthy granulations and new skin is growing from all margins.

The best results with these methods have been none too satisfactory for even when recovery occurs it is slow and tedious. Sulfonamides were



had a sudden sense of illness with abdominal distention generalized tenderness and symptoms and signs of rupture of the abscess. Another period of improvement followed and on the fifth postoperative day a large pelvic abscess was felt per rectum. The patient now complained of numbness in the right thigh and later a false emphysema was felt in Scarpa's triangle. Serum was used and gas was released by 4 incisions in the thigh the abductor muscles were found completely destroyed. The patient died shortly after the operation. Postmortem examination showed a perforated appendix containing a fecalith a peri appendiceal and a pelvic abscess and generalized peritonitis. *Cl. welchii* was cultured from the wound at operation as well as from the thigh postmortem. The author concluded that the spread of infection was presumably from the cecum to the abscess and from the iliacus muscle to the thigh. The fact that appendectomy was not performed makes this case of special interest.

#### PROGRESSIVE POSTOPERATIVE GANGRENA (PROGRESSIVE BACTERIAL SYNERGISTIC GANGRENE) OF THE ABDOMINAL WALL

A special type of progressive postoperative gangrene of the abdominal wall was first described by Cullen in 1924 and has since been extensively studied by Meleney. The condition is not frequent but a large number of the reported cases which according to Liedberg numbered 41 in 1936 followed appendectomy particularly appendectomy in which drainage was necessary. Meleney's studies indicate that the infection is due to the synergistic action of a microaerophilic non hemolytic streptococcus and a hemolytic *Staphylococcus aureus*.

The condition usually becomes evident at the end of the first or second week after operation either as an infection of the whole wound or as a localized induration about the retention sutures. The wound is at first red swollen and tender. Within a few days the margins of the wound or the suture perforations present a carbunculoid indurated appearance. The center of activity is purplish the outer zone a brilliant red. The whole region becomes exquisitely tender and this tenderness according to Meleney is the outstanding feature of the lesion. Within a few days the purplish areas become frankly gangrenous. The dead skin assumes a dirty grayish brown color and the surface is dull and like suede. The purple zone spreads outward into the red zone and the skin becomes swollen and elevated. The central aspect of the purple zone toward the zone of gangrene is sharply defined. On the outer side it fades off into the red zone which slowly advances in all directions.

## WOUND DEHISCENCE

For obvious reasons wound disruption is more frequent in an infected and therefore weakened wound than in an uninfected one and more frequent in drained than in undrained wounds since the drainage opening offers a prepared exit for protrusion of abdominal contents. It may occur in both old and young patients and in patients who present both good and bad risks but as the studies of Maes and his associates indicate the most serious ruptures and those attended by the highest mortality occur in patients who present bad risks especially when they are advanced in years.

Of the various theories advanced to explain wound disruption the most reasonable are the theory of hypoproteinemia which was demonstrated experimentally by Thompson, Rivdin and Frank and the theory of C-avitaminosis which was demonstrated by Linman and Ingalls. In the opinion of Rhoads and his associates a protein deficit of sufficient magnitude to retard wound healing seriously is more common than a C-avitaminosis. The formation of new cells such as fibroblasts which is part of the process of wound healing cannot occur without the aid of protein. If adequate protein cannot be supplied from dietary protein as it usually cannot be in seriously ill patients or from stores of labile protein or from other fixed proteins or the products of their catabolism all of which are uncertain sources it can readily be supplied in the emergency by transfusions of plasma or of whole blood and by the use of protein injections (p. 317).

Recent studies by Localio, Chassin and Hinton suggest that too much reliance must not be placed on serum protein values as an index of protein deficits. Their studies in 17 control cases, 21 cases of debilitating disease in which operation was performed and 17 cases in which post-operative evisceration occurred indicated that significant tissue protein depletion can exist with normal serum protein levels. The tissue protein concentrations in patients who suffered evisceration were significantly lower than those of the normal group of patients and the fascial protein concentrations were significantly lower in the patients who eviscerated than in the patients who were debilitated but who did not suffer disruption. On the other hand the serum protein levels in the patients who eviscerated were below normal in only 4 instances though in all cases the blood was drawn for study at the time of evisceration. The authors conclude that the determination of the existence of a protein deficit must be made from the estimate of the patient as a whole rather than

tested when they were introduced but produced no results. In 1945 however McIneny, Friedman and Harvey reported the use of penicillin in 3 cases of progressive bacterial synergistic gangrene, in 2 of which there was a prompt and complete response. In the third case results were also good but the response was less striking probably because the penicillin was inactivated by secondary contaminants. In view of this report, there would now be no justification for resorting to other measures in this condition until penicillin therapy had been given a fair trial. Streptomycin has not proved useful.

McIneny called attention to the fact that in most of the reported cases of progressive postoperative gangrene of the abdominal wall following acute appendicitis an appendiceal abscess had been drained and the wound had been closed by suture. He suggested on the concept that tissue tension in the presence of contamination might favor this type of infection that when an appendiceal abscess is drained the skin wound should be left unsutured as a prophylactic measure.

### TETANUS

Up to 1932 according to Kroger 39 cases of tetanus following appendectomy had been reported. To this number he added a case of his own following operation for gangrenous appendicitis and diffuse peritonitis. The stump was not inverted because of the induration of the cecum. Partial evisceration occurred on the tenth day and signs of tetanus developed on the nineteenth day. Immediate treatment with antitetanic serum was begun, the serum being introduced by the intramuscular, intravenous and intrathecal routes and sodium amytal, magnesium sulfate and morphine were also used. Death occurred 40 hours after the onset of symptoms from respiratory spastic paralysis. Culture and autopsy confirmed the diagnosis.

The author suggested that the normal occurrence of tetanus bacilli in the colon of many individuals is a possible argument for routine inversion of the stump following appendectomy. The idea that all patients submitted to operations on the appendix and colon be given a prophylactic dose of antitetanic serum he rightly dismissed as likely to lead to a higher mortality from serum reactions than from tetanus which is a rare complication of appendectomy and which should become even rarer as more and more of the population is immunized against it. Treatment is by the usual routine for tetanus. Penicillin can be employed tentatively but reported results with it in tetanus are highly dubious.

cavity is not contaminated. In one of the Charity Hospital cases a dissecting retroperitoneal abscess following perforation of the appendix had ruptured into the cecum. In another (not included in the tabulated series), rupture occurred into the ileum and partial intestinal obstruction followed.

*Case 21.* A 38 year old colored man had begun to have gradual cramping intermittent abdominal pain radiating to the lower back 72 hours before admission to the hospital. It subsided completely 15 hours before admission but 8 hours later following a blind meal suddenly recurred with the same characteristics and distribution. An hour later nausea and vomiting ensued. Purgatives had been taken daily after the onset of pain and had resulted in small bowel movements. The patient had lost 35 pounds during the past 4 months, his intake of food was small and he had drunk on an average of 2 quarts of wine daily. He had had two previous admissions to the hospital, the first 7 years before for chronic alcoholism and pellegry and the second 4 years before for acute edematous pancreatitis for which operation was undertaken on the mistaken diagnosis of ruptured peptic ulcer.

The patient was poorly nourished and developed. Physical findings were limited to vague slight generalized abdominal tenderness, moderate distention, tympanites on percussion and hyperperistalsis. rebound tenderness was lacking. The temperature was 96.8° F, the pulse 92, the respiration 28 and the blood pressure 114/82.

The red blood cell count was 4,300,000 and the white blood cell count 8,500 per cu. mm. with 68 per cent polymorphonuclear leukocytes. Erect plate of the abdomen showed multiple fluid levels and distended loops of small bowel (fig. 50 p. 200).

Operation was undertaken after 6 hours of parenteral hydration and constant intestinal decompression on a diagnosis of intestinal obstruction. The abdomen was opened under ethylene anesthesia through a left paramedian incision. The peritoneal cavity contained about 200 cc. of cloudy fluid. The small intestine was greatly distended. The omentum was bound down in the region of the terminal ileum. When it was released it could be seen that the appendix had ruptured into the ileum 3 feet from the ileocecal junction. There were old, well organized adhesions about the site of the fistula. The ileum was distended proximal to this point and collapsed distal to it. The appendix was removed by the retrograde technique after which the opening into the ileum was closed transversely, the suture line being protected by a free omental graft. Sulfanilamide (5 gm.) was placed in the peritoneal cavity before closure. Recovery was uneventful.

Vesico appendiceal fistulas are infrequent and are not likely to occur when the cecum is in the normal anatomic position unless the appendix is unusually long. Twenty seven of 592 vesico intestinal fistulas reported

on the basis of laboratory tests. They also stress the importance of their observation that the lowest tissue protein values were in the fascial layer in view of the importance of this layer in wound healing.

The studies of Birtlett and his associates on human wounds are in agreement with the opinion of Rhoads and his associates that Cavitaminosis is a possible but not a frequent cause of wound disruption. These workers showed (1) that the average person under ordinary conditions of diet and absorption has a high enough ascorbic acid content in the tissues to permit normal wound healing, (2) that in the occasional case a marked depletion of vitamin C as shown by a low plasma ascorbic acid may interfere with normal wound healing as shown by tests of tensile strength and determinations of tissue ascorbic acid content and (3) that normal wound healing can be encouraged by the use of vitamin C therapy.

The use of nonabsorbible suture material whatever be the surgeon's general preference, is clearly a wise precaution in cases in which disruption is a possibility though it is not an infallible protection. Cotton silk and silver wire are all useful in this connection. A surgeon who practices early ambulation should always use nonabsorbible suture material. Complicated methods of closing have never proved useful in the prophylaxis of wound dehiscence.

### CECAL FISTULAS

*Internal Fistulas* Petty in 1933 seems to have been the first to call attention to the great danger of internal fecal fistulas following appendectomy. The accident may occur as the result of vascular thrombosis and infection of the cecal wall or as the result of the destruction of the blood supply of the cecum from tight pursestring sutures. It may also follow injudicious postoperative treatment including the use of pitressin and similar agents, the early resort to purgation or the use of too large quantities of water in enemas. Any of these practices may literally blow the ligature off the appendiceal stump or cause the pursestring suture to give way.

Failure to detect the accident is almost inevitably fatal. If it is recognized the proper treatment is the conversion of the internal fistula into the external variety with later appropriate treatment of the external fistula.

The rupture of a diseased appendix into some one of the abdominal viscera such as the rectum or the bladder creates an internal fecal fistula but the consequences are less disastrous because the peritoneal

portion of the fecal stream passes out through it but occasionally the major amount of feces is excreted by this channel. Theoretically it should not be the cause of symptoms other than inconvenience and social embarrassment. Actually a patient with a fecal fistula is often debilitated, emaciated, and anemic. The skin may be excoriated and the adjacent tissues inflamed.

The mere colonic odor of a discharge is not sufficient to establish the diagnosis of fecal fistula. *B. coli* infections are very frequent in complicated acute appendicitis. The passage of gas or the demonstration of feces in the discharge is conclusive. In doubtful cases the diagnosis may be established by the use of dye by mouth, by roentgenology with a barium meal, or by the injection of an opaque substance into the fistulous opening. The latter method should always be employed before corrective operation is undertaken to determine the presence or absence of obstruction in the distal loop.

If a fecal fistula, whether spontaneous or surgical in origin, does not close of itself within three months at the outside, the presence of a foreign body, such as a fecolith, a sponge, or an unabsorbed suture, should be suspected. If only a drainage operation has been performed, the appendix itself may be assumed to be the origin of the fistula. Fitz described such a case in his first paper.

A patient had a fecal fistula in the groin following the incision of a tender swelling in this region. Healing did not occur, and it was observed that pressure over the cecum caused intestinal contents to appear in the wound. After an operation undertaken to promote healing of the sinus, about an inch of perforated appendix protruded from the wound. A similar protrusion had been observed six months earlier, but its significance had apparently not been appreciated.

A foreign body or the appendix which is responsible for a fistula should be removed at once. Otherwise it is well to give the fistula a chance to close itself by the use of conservative measures. These include the elimination of infection, the use of silver nitrate to prevent epithelization, plugging of the opening by gauze, iodoform packs, chewing gum, or mechanical devices, or pressure with a sponge and adhesive tape. Royster several times secured permanent closure by the use of tapes soaked in compound tincture of benzoin and packed deep into the opening. Supplemental measures include the use of a low residue, high caloric diet, the correction of disturbances resulting from loss of fluids, electrolytes, or enzymes, the correction of nutritional deficiencies, and the use of chemotherapeutic and antibiotic agents.

by Kellogg were of appendiceal origin as were 2 of 31 cases reported by Barnes and Hill Pemberton Pool and Miller reported 5 cases observed at the Mayo Clinic

The most usual manifestations of a vesico appendiceal fistula are burning and frequency of urination pyuria possibly gross hematuria and pneumaturia feces sometimes appear in the urine Associated with the cystitis which is intractable may be suprapubic discomfort or pain rectal pain temperature elevations and chills Cystoscopy reveals in early cases an area of acute inflammation and edema in the fundus of the bladder on the right side and in late cases an area of granulation tissue the fistulous opening can sometimes be detected in the center of the affected area The remainder of the bladder wall is usually entirely normal The diagnosis can sometimes be confirmed by injection into the bladder of a radiopaque solution which enters the colon through the fistula but Barnes and Hill found the method undesirable

The treatment of choice is appendectomy and closure of the bladder opening by a two stage procedure if necessary supplemented by catheter drainage of the bladder and by appropriate use of chemotherapeutic and antibiotic methods That the condition is potentially serious is shown by the fact that in the 5 cases reported by Pemberton and his associates there were 2 deaths

*External Fistulas* Although external fistulas may occur after any abdominal operation they are particularly frequent following appendectomy In the 155 cases reported by Mayo and Schlicke 50 followed appendicitis but Dixon and Deutermanns estimate that 4 per cent of all cases of appendicitis are followed by fistula formation seems unduly high Only 42 were recorded in 6441 surgical cases at the New Orleans Charity Hospital and it seems doubtful that a complication of such gravity would not be noted in practically every case in which it occurred

The causes of fistula formation vary The bowel may be injured during operation The cecum or ileum may be secondarily involved in the disease process or necrosis may be caused by the use of tight sutures Fistulas are frequent following inadequate drainage of an abscess or following a drainage operation in which the appendix has not been removed When cecostomy or appendiceal cecostomy was a popular method of treating appendicitis with rupture and peritonitis a fecal fistula was deliberately created which sometimes had to be closed by a surgical procedure Fistula formation is also likely to occur in debilitated or tuberculous patients in whom healing is slow

A true fecal fistula which must be distinguished from a sinus tract is really an abdominal anus Usually the fistula is small and only a minor

than they run without surgery. As a matter of fact patients with serious organic disease always live in jeopardy no matter what is or is not done to them and it seems logical to assume that the correction of a possibly lethal disease subjects them to less risk than permitting the disease to continue unchecked. Furthermore many patients who die of heart disease after appendectomy die because they were permitted to reach a serious or even moribund state before their disease was diagnosed. Cardiac disease is not a common cause of death in properly managed surgical patients.

This does not mean of course that surgery is free from risk. It is not. Every effort should be made to shorten the duration and minimize the trauma of anesthesia and operation. Prophylactic measures to prevent such complications as shock, peritonitis and pneumonia should be particularly thorough especially in patients advanced in years. Bed rest seems to have nothing to do with either the incidence or the mortality of coronary occlusion after operation. Renal disease however adds considerably to the cardiac risk. In the 494 operations performed on 414 cardiac subjects in the series reported by Butler and his associates the mortality without nephritis was 4.9 per cent and with nephritis 14.8 per cent. There were no deaths in 15 appendectomies.

*Infusions must be given slowly and cautiously in patients with cardiac and cardiorenal disease and it may be necessary to administer smaller quantities than would be desirable under other circumstances in order not to overtax the circulation. Sodium chloride should also be administered cautiously and perhaps in smaller quantities than would otherwise be indicated because of the risk of edema from retention (p. 311).*

## RENAL DISEASE

Renal disease needs no extended discussion since it does not differ from similar disease in the usual circumstances and after any operation. Urinalysis should be carried out before operation in all cases and kidney function tests should be performed as indicated to identify renal disease and dysfunction. A patient in whom renal disturbances are discovered should immediately be placed in the charge of a competent internist. As has been pointed out modifications in replacement therapy may be necessary to meet special conditions in the individual case. The mortality in properly managed cases is not unduly high in the absence of other complications but it is considerably higher as has just been noted when renal and cardiac disease are associated.



If these measures are not successful within a reasonable period of time surgery should be instituted without further delay. Preparation for operation includes intensive preparation of the intestinal tract by the use of a nonresidue diet and saline laxatives. Sulfathiazidine is given by mouth in dosages of 4 tablets (77 gr each) every 4 hours for 5 days. Pre operative intraperitoneal vaccination was never widely popular and is no longer necessary since effective chemotherapeutic agents are available.

Each case must be handled according to the special indications which exist; no general plan of procedure is possible other than strict observance of the rule that an intraperitoneal procedure should not be undertaken if an extraperitoneal operation can be done with equally good prospects of success. Occasionally all that is needed is inversion of the mucosa of the bowel after mobilization and closure of the skin but as a rule simple operations are attended with a high proportion of unsuccessful results. Suture of the intestinal wall and plastic and short circuiting operations are frequently necessary but resection is seldom indicated unless the fistula impinges on the ileocecal valve.

Postoperative treatment follows the usual routine described for appendectomy for advanced disease (p. 298) and includes chemotherapy and replacement therapy. Nonresidue fluids are permitted by mouth in small graded amounts within 24 hours after operation. By the fifth day in the usual case the patient is taking a full nonresidue diet and the ordinary diet is resumed after the twelfth day.

Fistulas should not be regarded as simple conditions nor should operations to repair them be regarded as free from danger. The mortality in the 41 postappendicitis cases in which closure was carried out at the Mayo Clinic was 18 per cent and the known mortality in the 49 fistulas following appendicitis reported by Lewis and Penick from the Johns Hopkins Hospital was more than 8 per cent.

#### CARDIAC AND CARDIOVASCULAR COMPLICATIONS

Cardiac complications encountered in patients with acute appendicitis either before or after operation do not differ from similar diseases encountered under other circumstances. Generally speaking the risks of surgery in cardiac subjects are greatly exaggerated. Patients with functional cardiac disease run practically no risks to which the patient without such disease is not also submitted and patients with serious disease run far less risk from an appendectomy performed without delay.

The most important factor in the production of postoperative pulmonary complications is therefore the state of hypoventilation which occurs during anesthesia and which is likely to continue for some days thereafter especially if unnecessarily tight dressings are used.

Although some surgeons believe that the duration of the operation makes no difference others do not accept this point of view. The same difference of opinion exists as to the importance of the depth of anesthesia. Careful studies usually show that both the incidence and the mortality of postoperative pulmonary complications increase in proportion to the depth and duration of the anesthetic. The influence of anesthesia is seen in the higher male incidence of postoperative pulmonary complications. As Burford expresses it for all practical purposes the induction of a premedicated male is approximately as difficult as the induction of an unpremedicated female.

The pre-operative state of the patient is naturally a factor in the production of pulmonary complications. This refers not only to pre-existing respiratory complications in spite of which an anesthetic must be given in an emergency but also to the general physical state as expressed in terms of physiologic rather than of actual years. All studies show that very young and very old patients and debilitated patients especially when infection is present are peculiarly prone to develop respiratory complications after operation. In the 6441 patients operated on at the New Orleans Charity Hospital 11 of the 18 fatal pulmonary complications occurred in individuals over 40 years of age who accounted for only 12.2 per cent (778) of the cases. Taylor and his associates who studied postoperative pulmonary complications in relation to pre-operative estimates of risk found an almost uncanny degree of correlation between these two factors. That operation need not be deferred because of existing pre-operative complications however is evident from Collins' statistics which showed three times as high a mortality with conservative therapy under these circumstances as with operation under a skillfully given anesthetic. There were no deaths among the 7 patients at the New Orleans Charity Hospital who were operated on in the course of pneumonia.

*Clinical Picture* In the majority of cases postoperative pneumonia does not differ from pneumonia observed at other times. It is likely to be of the bronchopneumonic rather than the lobar type. Most often the infection is at the bases of the lungs where the results of prolonged decubitus especially in older subjects show as hypostatic congestion and atelectasis.

In the massive variety of atelectasis the picture may be one of cys-

## PULMONARY COMPLICATIONS

*Etiology* Anesthesia itself barring the occasional improperly given anesthetic is no longer regarded as the chief cause of postoperative pulmonary complications. That statement can be proved by two facts (1) Pulmonary complications are at least as frequent after spinal as after inhalation anesthesia (2) The inhalation of an irritating gas causes edema which is practically never an immediate postanesthetic complication and not atelectasis which in some degree is the chief immediate finding in practically all cases of postoperative pulmonary disease.

The most important cause of pulmonary complications after operation is the postoperative reduction in the vital capacity of the lungs which ranges from 20 to 70 per cent. Wendell Henderson has pointed out that roentgenologic examination of the chest after operation practically always shows a state of hypoventilation the diaphragm being removed upward from its normal position much further than it ever moves under the deepest and most forcible voluntary expiration. The fundamental reason for the displacement is a general decrease of body tonus affecting all the muscles of the body including the respiratory muscles and the diaphragm. Spinal analgesia in itself abolishes the tonus in the parts of the body anesthetized though the high analgesia which produces intercostal paralysis is not necessary in the usual operation for appendicitis.

The result of this loss of tonus is that many minute areas of degeneration are produced in the lungs and amount to a state of multiple atelectasis a miniature reproduction of the condition seen in the more infrequent massive atelectasis or pulmonary collapse. The next consequence is the accumulation of mucus in the airways sometimes to such a degree that one or more of the major bronchi are completely blocked. If the occlusion lasts for any length of time the gases in the occluded areas are absorbed into the blood and these areas collapse. If the collapse involves extensive areas there is a mediastinal shift to the affected side.

If deep breathing could be resumed promptly after operation the state of affairs described could not become serious. The airways would be kept open, mucous collections would be coughed up and a normal respiratory state would promptly be restored. But deep breathing after abdominal surgery even when only the lower abdomen is involved is seldom resumed voluntarily for several days. The reason reduced to its simplest terms is that deep breathing hurts. The incision naturally is painful and the pain is increased when deep breathing is practiced.

the bed and observation should be maintained to see that he does so. In complicated cases the position should be changed at intervals of 2 hours or less by the nurse until the patient can move himself about.

In addition to changes of position the patient should be instructed to take several deep breaths at hourly intervals the incision being splinted by the nurse with the palms of her hands if deep respirations are painful. Prophylactic measures should be redoubled for the patient who a day or two after operation develops fever, cough and a purulent sputum for he is a candidate for more serious trouble unless steps are taken to prevent it.

As soon as possible—which can be within a few hours of operation—the patient may be placed in the semi sitting position. Whether or not the surgeon is an advocate of immediate postoperative ambulation the measures described will prevent most cases of postoperative pneumonia.

The prophylactic use of inhalations of carbon dioxide has proved disappointing but the routine use of oxygen therapy at the conclusion of operation and if necessary after operation has given excellent results. Graham and Brown suggested a simple method of administering it when an inhalation anesthetic has not been used by making a tent of the end of the laparotomy sheet and running a stream of oxygen into it.

*Therapy.* Postoperative atelectasis should be treated without delay by bronchoscopic aspiration by a competent bronchoscopist. Postoperative pneumonia should be treated just as pneumonia after other circumstances that is by a competent internist with chemotherapy, antibiotic therapy, serotherapy and supportive measures. Empyema, pulmonary abscess and pulmonary gangrene should they develop should also be treated as they would be ordinarily.

### PHLEBOTHIROMBOSIS AND THROMBOPHLEBITIS

Vascular complications are considerably less frequent after appendectomy than they are after a number of other procedures but they are potentially just as serious. As under other circumstances phlebothrombosis may be followed by fatal embolism and thrombophlebitis may terminate in considerable and often permanent disability.

*Pathologic Process.* The distinction between phlebothrombosis and thrombophlebitis is extremely important. Phlebothrombosis (Ochsner and DeBakey) or bland or quiet thrombosis (Homans) is a noninflammatory vascular lesion in which the predisposing factor is increased

trophe. The onset usually occurs within 48 hours of operation less often later with a sudden elevation in temperature, pulse and respiration. The patient becomes dyspnoic and sometimes cyanotic. He makes futile efforts to cough. He complains of severe pain in the chest or upper abdomen. His expression is anxious. In milder cases the symptoms are of the same character but less severe and the condition may be entirely overlooked since the areas of involvement are scattered and limited unless repeated examinations are carried out including roentgenologic studies. To the use of the X-ray is due the present concept that many postoperative complications formerly diagnosed as pneumonia are actually instances of atelectasis.

The variability of the chest findings in postoperative atelectasis is the outstanding physical characteristic of the condition. In the severe case the most prominent finding is a shift of the heart and the mediastinal structures to the affected side. Expansion over the affected area may be limited. Often there is retraction of the interspaces on the affected side with increased expansion on the opposite side. If the atelectasis persists after the causative bronchial obstruction has been relieved signs of consolidation are apparent. While cyanosis may be due to mechanical interference with breathing it should always be regarded as a possible indication of impending pneumonia especially in older persons.

*Prophylaxis.* Prophylactic measures will usually prevent postoperative pulmonary complications. Pre-operative sedation should be given sufficiently in advance of operation for the attainment of the maximum effect during induction of the anesthesia so that it will begin to wear off as anesthesia progresses. Anesthesia should be induced in as quiet circumstances as possible with the transition from induction to deep anesthesia gradual and deliberate. The anesthetist should never be hurried. It should be maintained on the happy level on which moderate depth and deep relaxation meet. The suction apparatus should be used to clear the bronchial passages preferably before the patient's breathing indicates that it is necessary and should be used routinely at the conclusion of the operation.

The most satisfactory method of postoperative prophylaxis is the simple one of frequent changes of position. The patient should not be permitted to lie constantly in any one position especially on the back. In uncomplicated cases he can be turned on his side immediately after operation to facilitate the gravity flow of pulmonary secretions to maintain the cough reflex and to guard against the aspiration of vomitus. As soon as consciousness returns he can move himself about

F on the second postoperative day and the highest pulse rate was 96. On the sixth day when he was afebrile the patient complained of pain in the left leg which rapidly became edematous and then of weakness. The pulse became weak and irregular and death occurred within 2 hours apparently from embolism.

Postmortem examination revealed a pulmonary embolism with three thrombi at the bifurcation of the pulmonary artery, thrombosis of the left common iliac vein and parenchymatous degeneration of the liver.

The liver damage in this young man in the absence of peritoneal infection or other obvious causes is worth noting (p. 253).

*Clinical Picture and Diagnosis.* In phlebothrombosis symptoms are practically nonexistent and signs are slight. Evidence of the existence of the condition must be sought for and since they develop both promptly and insidiously they must be looked for carefully and often preferably twice daily. They amount to little more than slight swelling of the ankle, slight distention of the superficial veins of the foot and minor discomfort on palpation of the calf muscles. It is important to make regular attempts to elicit Homans' sign (pain in the calf on forcible dorsiflexion of the ankle) and to make comparative studies of both legs particularly when the patient complains of unilateral pain or tenderness in the calf to detect the presence of slight swellings.

Allen and his associates among others have laid considerable emphasis on simultaneous elevation of the temperature, pulse and respiration as a warning of an impending vascular accident but DeBakey in a study of 332 cases of thromboembolism over a 5 year period at Charity Hospital of Louisiana at New Orleans noted that in a third or more of all cases neither this warning nor any other suggested the possible development of the condition.

Thrombophlebitis is unlikely to remain unrecognized very long because the symptoms and signs are clear cut: pain, tenderness along the course of the involved vein and swelling of the extremity which may take the form of true phlegmasia alba dolens. Ochsner called attention to the significance of the paradox of a patient suffering from fever with an elevated bodily temperature elsewhere whose extremity is white and cold; the explanation is that the irritative lesion within the vein has produced a concomitant spasm of the arterioles.

*Prophylaxis.* Welch and Faxon have properly pointed out that although phlebitis is often regarded simply as a nuisance and embolism as an act of Providence neither point of view is justified. It is the surgeon's duty so far as he can to identify patients with bland thromboses who are candidates for fatal emboli and to adopt measures that

coagulability of the blood brought about by tissue damage. The precipitating factor is circulatory stasis which determines the site of the thrombus. The clot is of the red or coagulation type; its attachment to the wall of the vein is precarious and pulmonary infarction and embolism are always possibilities. Thrombophlebitis on the other hand is an inflammatory lesion. The clot is of the white or mixed type; it is firmly attached to the wall of the vein and embolism is a possibility only when which is not usual it suppurates and liquefies.

All the evidence indicates that phlebothrombosis originates in the deep veins of the leg usually in the muscles of the calf and progresses upward through the adductor region to involve the femoral and then the iliac vessels. In some cases the opposite leg is involved in what on the surface at least seems to be a retrograde fashion. The importance of thrombi in the veins of the leg is less appreciated than it should be because these veins are not investigated routinely at autopsy. When ever they are examined the evidence is there. Of 280 cases of pulmonary embolism without other signs of thrombosis (collected by Zilliacus and discussed by Jorpes) in Swedish clinics 114 were immediately fatal. In 60 of the 86 cases in which autopsy was carried out thrombi were found deep in the veins of the leg. Jorpes' conclusion seems justified that by proper investigation many of these cases could have been diagnosed antemortem in time to save the patients' lives.

Small emboli because they give rise to no symptoms or to vague symptoms are probably much commoner after operation than is generally believed. Massive embolism on the other hand is perhaps not as common as is thought. In the absence of postmortem examination the diagnosis is sometimes made too readily as a convenient method of explaining unexpected deaths. Be this as it may, there is no more dramatic happening and no greater tragedy than the embolism that occurs literally like a bolt from the blue in a patient whose recovery has been completely uncomplicated as in the following case.

**Case 22.** A colored man 27 years of age had been ill for 4 days with generalized abdominal pain and nausea and vomiting. Physical examination revealed slight generalized abdominal rigidity and generalized tenderness. The temperature was 100° F, the pulse rate 80 and the respiratory rate 22 per minute. The white blood cell count was 12,250 per cu mm with 83 per cent polymorphonuclear leukocytes.

A diagnosis of subsiding acute appendicitis was made and appendectomy was performed 24 hours after his admission to the hospital through a right rectus incision under spinal analgesia. The appendix was gangrenous.

The patient made a smooth recovery. The highest temperature was 101.3°

monary embolism 75 of which were fatal. The incidence in appendectomy was 1:1300. Half of the patients who died of embolism lived less than 10 minutes after the condition became manifest, three quarters less than 30 minutes, and only 10 per cent more than an hour. Between 1939 and the date of the report (1946) 3 patients died of embolism after appendectomy. If treatment had been undertaken routinely to avert these catastrophes, it would have been necessary to heparinize or dicoumarolize 3900 patients and the hemorrhagic complications, as Linn and Hooker pointed out, would undoubtedly have been notable. They added that the plan was even less attractive when it was remembered that 1 of the 3 patients who died of embolism following appendectomy was under treatment with heparin when he had a second embolus which proved fatal 7 days after the first.

*Therapy.* The treatment of thrombophlebitis is comparatively simple. In view of the spastic etiology of the lesion, the rational treatment is vasodilatation, which is best secured by sympathetic block, repeated as often as necessary. Ochsner and DeBakey recommend the injection of 5 cc. of 1 per cent procaine solution through four needles at points two fingerbreadths lateral to the spinous processes of the first, second, third, and fourth lumbar vertebrae. Compression bandages are applied from the toes to the groin, the muscles are contracted against resistance, and ambulation is practiced as soon as the fever subsides. Under this type of therapy edema and pain rapidly disappear, in contrast to older methods of treatment which required long periods of hospitalization.

When a patient develops any evidence whatsoever of phlebothrombosis, immediate active therapy is necessary. The rationale of venous ligation is twofold: to prevent the clot from progressing proximally, and extending into the deep venous system, and to prevent pulmonary embolism. The technique recommended by Allen and his associates is simple and satisfactory. The junction of the superficial and profunda femoris veins is visualized and the superficial femoral vein is tied at this point. There is thus no blind segment of vein distal to the site at which these two veins unite to form the common femoral vein. If there is reason to believe that the thrombus has extended higher, the iliac vein can be ligated above the presumed site. The fact that thrombosis may have occurred far in advance of the area indicated by the clinical manifestations has led some observers to advocate ligation of the inferior vena cava. It seems more reasonable to reserve this procedure for very carefully selected cases, usually those in which sepsis is a factor, as it is not in the ordinary case of phlebothrombosis.



will prevent phlebothrombosis. Since appendectomy for acute appendicitis is an emergency procedure the discussion of specific pre operative preventive measures is merely academic with the possible exception of the use of compression bandages extending from the toes to the groin as recommended by Ochsner and DeBakey in all patients over 40 years of age who are submitted to surgery.

The most effective form of postoperative prevention is free and active movement of the legs with active forcible bending and flexing of the foot against resistance. Such movements are practical in bed even for a patient who has just been operated on. Adams reported that at the Lahey Clinic where instruction for energetic postoperative leg exercises are distributed to the patients in all languages the adoption of this simple measure has reduced the incidence of thrombo embolic phenomena by 50 per cent.

Fowler's position is to be avoided. It introduces compression in the popliteal space and discourages movement. Flewkes attributed the alarming increase in the incidence of embolism at the Toronto General Hospital after 1931 to the fact that Gatch beds had been supplied in all wards and that the patients as a result breathed less deeply, flexed their knees with no effort and were too comfortable to move. Deep breathing favors the return flow of blood through the venous system to the lungs.

Early ambulation has not entirely prevented thrombosis and embolism one reason possibly being as Ochsner pointed out in a discussion of Allen's 1946 contribution before the American Medical Association that the term has been interpreted by some physicians to mean permitting the patient to sit up in a chair. The result is not movement but compression of the popliteal space against the edge of the chair with the same undesirable results as when Fowler's position is used.

It is open to question whether mass prophylaxis is justifiable even in patients 50 years of age and over in whom vascular complications are most frequent. Allen and his associates take the position that it is. At the 1946 Clinical Congress of the American College of Surgeons Allen reported on 1518 ligations of the femoral vein 453 of which were prophylactic. Others have suggested mass prophylaxis with dicoumarol but the idea does not seem practical. As DeBakey pointed out it would mean an intolerable increase in medical and nursing care and would undoubtedly cause more deaths from hemorrhage than it would avert from embolism in a condition in which the expected incidence of the complication is 1 in 500 and the actual incidence much lower.

Lam and Hooker took the same position. At the Henry Ford Hospital over the 21 year period ending in 1944 there were 280 instances of pul

hepatic disease hypertension hyperthyroidism diabetes and of course the hemorrhagic diathesis

Anticoagulant therapy is rather promptly effective when heparin is used and seems particularly indicated in patients with pulmonary embolism who are so ill that even the minimal manipulation of venous ligation might be intolerable. It must be remembered however that anticoagulant drugs have no effect on emboli which have already formed. Their action is to prevent further showers of emboli. Their use should be supplemented by oxygen in high concentration to minimize respiratory and cardiac efforts because the oxygenation of these patients is greatly impaired and voluntary deep breathing is undesirable. Ochsner and DeBakey have suggested novocain block of the cervicodorsal sympathetic ganglia to control the spasm probably present in the remaining pulmonary vessels. It has been suggested that heparin and dicoumarol should be discontinued gradually rather than abruptly as deaths from embolism have occurred when one or the other has been abruptly discontinued.

Anticoagulants must be used with the greatest care in patients who have been operated on (or who are to be operated on) because of the danger of initiating hemorrhage into the wound or the operative field. The danger is minimized by extreme care in hemostasis at operation and by absolute control by laboratory studies of the clotting and prothrombin times.

Zilliacus material (reported by Jorpes) supplements Murray's previously cited figures. There were no deaths in 103 patients with sudden pulmonary embolism who survived long enough for treatment and who were treated by heparin or dicoumarol against 21 deaths in 63 patients treated without these agents. In 900 cases of thrombosis or pulmonary infarction treated by Zilliacus and Brier (reported by Jorpes) with heparin or dicoumarol or both there were 6 deaths 0.67 per cent against 88 deaths 16 per cent in 543 cases treated without these agents.

#### MISCELLANEOUS POSTOPERATIVE COMPLICATIONS

*Hemorrhage* If as sometimes happens the appendix must be detached from the iliac vein or artery the vessel must be carefully examined to be certain that it is not involved in a necrotic process and that it has not been damaged by the manipulations necessary to detach the appendix. Fitz in his first communication collected 3 cases of this kind from the literature 2 of which involved the iliac artery

If it seems likely that the thrombus has extended above the junction of the superficial and profunda femoris veins some surgeons instead of ligating the iliac vein prefer to open the femoral vein at its junction with the sphenous vein and remove the thrombus by suction free retrograde flow of blood from the proximal end of the vein indicates that it has been completely removed. The femoral vein is then ligated. Thrombectomy is a practical and often a lifesaving procedure. Embolectomy on the other hand has been performed in only a few instances and the nature of the pathologic process militates against a high degree of success. Moreover the promptness with which it must be carried out if life is to be saved usually makes it entirely impractical even if means for its performance are constantly on hand.

Anticoagulant therapy should be employed following venous ligation or is a primary method of treatment in all patients who have survived pulmonary embolism because a certain proportion estimated at about 1 in every 5 is likely to develop another or more than one other which may be fatal. Murray reported 149 cases in which he personally supervised the administration of the anticoagulant and in which there were no deaths although 52 patients when they were first seen by him presented all the serious and dreadful effects of massive pulmonary embolism. Four patients developed evidence of additional emboli while they were under treatment. This is a brilliant record made more brilliant by the fact that in a fairly large number of similar cases which he saw in consultation but in which he did not control the treatment there were 3 deaths.

If heparin is used Murray pointed out it must be carried to the point where the clotting time of the patient is kept at or about 15 minutes. Frequent determinations are required for the effect of heparin is evanescent and the dosage depends upon the individual response. The drug must be continued until the healing process in the thrombotic area has reached the stage at which no further thrombosis will occur. In patients who can be ambulatory within 3 or 4 days treatment is necessary for 7 to 9 days. If bed rest is required treatment must be continued for 3 or 4 weeks.

Heparin is expensive and is not convenient to administer even in the form of Loewer's heparin Pitkin menstruum. It is therefore wise to use dicoumarol which can be given by mouth as a substitute for it. Heparin however must be given for at least 48 hours since dicoumarol does not become effective for that length of time or longer. Dicoumarol must be used with the same precautions as heparin. It is contraindicated in

extremely infrequent the third explanation seems most reasonable. The liver damage in view of the youth of the patients is interesting.

**Hematemesis** Hematemesis has occasionally been reported after appendectomy. Collected cases (which now number about 40) show that after a single hemorrhage recovery is usual but that repeated hemorrhages have a more serious prognosis. Dieulafoy explained the phenomenon on the basis of toxic absorption. Kitchen who reviewed the literature reported a personal case following appendectomy and drainage for gangrene and perforation of a retrocecal appendix 30 hours after the onset of the illness. A purgative had been taken. Death occurred on the seventh day after two hematemeses. Postmortem examination revealed a purulent peritonitis, diffuse thrombophlebitis of the omental vessels and multiple erosions of the gastric mucosa. The liver and portal vein were not involved. In the apparently similar case (a 6 year old child with acute ulcerative appendicitis) reported by John postmortem examination showed death to be due to a chronic duodenal ulcer which had eroded the gastroduodenal artery, the floor of the ulcer was formed by the pancreas.

**Parotitis** Infection of the parotid gland usually is ascending by way of Stenson's duct. The symptoms are local heat, redness and swelling. The condition is most often observed in patients with low resistance, oral sepsis and dry mouths. The incidence is highest in dehydrated subjects.

The best treatment is prophylaxis consisting of oral cleanliness and preservation of the fluid balance so that the mouth is always moist. The use of chewing gum and of hard candy which increases the flow of saliva is both prophylactic and therapeutic. Mouth washes are also useful. Surgical drainage may be necessary if simple measures such as hot compresses, aspiration and gentle irrigations do not prove adequate. Kelly and Dowell and Pendergrass and Hodes have reported excellent results with small doses of X ray.

**Hepatic Complications** Numerous observers of whom I am one have pointed out the postoperative depression of liver function which occurs after all types of abdominal surgery and all varieties of anesthesia and have also pointed out the unexpectedly high incidence of latent and subclinical hepatic dysfunction in the general population (p. 247). Whether consciously or unconsciously physicians who employ replacement therapy on the proper indications are guarding against postoperative complications due to liver damage and these precautions are adequate in most cases.

Severe liver damage was evident at necropsy in 3 of the fatal

The vein is more likely to be involved in a necrotic process than the artery because its coats are thinner and the blood circulates less rapidly in venous channels. Thrombophlebitis however is a more usual sequel than either necrosis or hemorrhage. If the vein on examination proves to be damaged it should be reinforced by peritoneal flaps.

Ottolenghi reported an instance of delayed hemorrhage from this cause.

A 7 year old boy 3 weeks after the removal of a gangrenous appendix had a hemorrhage from the wound. It was controlled by tamponade and a transfusion was given. Five days later another and more serious hemorrhage necessitated reopening the abdomen. The external iliac vein and artery both of which were ulcerated were ligated and the child recovered after repeated transfusions. Dry gangrene developed in the leg on the affected side.

If the appendix is adherent to other abdominal structures particularly the intestine it is usually well to invert the point of attachment with Lambert sutures to guard against hemorrhage as well as perforation.

*Hemorrhagic Diathesis* In the only instance of a possible hemorrhagic diathesis in the Christy Hospital series bleeding lasted for 24 hours and no real cause was ever found for it. The serum volume index (Boyce) was within normal range operation was performed without difficulty and recovery thereafter was smooth.

Simeone and Stewart reported 2 cases of the hemorrhagic diathesis in acute appendicitis with perforation and generalized peritonitis.

One patient a 17 year old male began to bleed from the wound on the twelfth day following the appearance of jaundice on the second day. Two days later the serum bilirubin of the blood was 23 mg per cent and the non protein nitrogen was 43 mg per cent. At autopsy a diffuse central necrosis of the liver was found with a hemorrhagic exudate in the retroperitoneal space. The second patient a 13 year old girl developed jaundice on the eighth day and began to bleed from the wound on the thirteenth day. On the fifteenth day the plasma prothrombin was 38 per cent of normal and the nonprotein nitrogen of the blood was 38 mg per cent. Vitamin K and bile acids were administered but death occurred the same day. The first patient was operated on under spinal analgesia and the second under ether anesthesia.

The authors offered three possible reasons for the hemorrhagic diathesis in these cases. (1) The diet was deficient in vitamin K. (2) The absorption of vitamin K from the intestinal tract was defective. (3) The liver function was disturbed. Since the first two causes are common and the hemorrhagic diathesis in acute appendicitis is

The patient vomited almost continuously following operation and suffered from considerable abdominal distention. The temperature was 104° F and the pulse rate 140. Standard treatment for peritonitis was instituted. The patient voided voluntarily once following operation and thereafter catheterization secured only a few cubic centimeters of urine which revealed nothing abnormal on examination. On the fourth postoperative day the patient was cold and clammy, the pulse was thready and there was considerable upper abdominal distention. He was irrational and the blood pressure was 0/0. The urea of the blood was 105.7 mg per cent. There was no response to infusions, transfusions, stimulants and other measures and death occurred on the fifth postoperative day.

Postmortem examination revealed a clean peritoneal cavity. The intestines were moderately distended. The liver which weighed 900 gm showed gross and microscopic degenerative changes and fatty metamorphosis. The kidneys showed congestion of the glomerular tufts.

Attention has been called elsewhere (p 326) to the possible production of liver damage and the possible increase of pre-existent liver damage in patients treated by chemotherapy.

*Miscellaneous Complications.* Almost any conceivable complication may occur after operation for any variety of appendicitis though one condition is not necessarily related to the other. In the Charity Hospital series for instance a number of children developed chickenpox, mumps and measles during convalescence but the chronology suggests that the majority were operated on during the incubation period. Psychiatric disturbances are occasionally observed but as a rule are transient.

## DELAYED COMPLICATIONS

Of the delayed complications of acute appendicitis the most important are fecal fistula which has already been discussed (p 236) and hernia. Hernia may be due to a number of causes including the cutting of the nerve supply of the muscles, the division of muscle fibers, prolonged drainage and wound infection with or without partial evisceration. Contrary to the usual belief hernia may occur after any type of incision including the McBurney.

It is highly doubtful other things being equal that the length of the incision has anything to do with the development of hernia provided that the peritoneum and aponeurotic layers are accurately approximated when closure is done and that the peritoneum is sutured so as to bring fairly wide areas of serous tissue into contact with each other and so as to leave no intraperitoneal protrusions to give rise to adhe-

surgical cases at the New Orleans Charity Hospital. Two of the histories are appended. In the first (case 23) the pathologist considered the condition acute yellow atrophy of the liver with the severe wound infection incidental. In the second case (case 24) the patient seems to have died a so called liver kidney death. The third death in this group was also of the liver type. Cases of this type are of course distinct from cases in which pyelophlebitis and liver abscess furnish an obvious cause for the liver disease.

*Case 23.* A white woman 50 years of age developed epigastric pain, nausea and vomiting after her evening meal 48 hours before admission to the hospital. The pain radiated to the lower abdomen and the right lower quadrant. The temperature was 102.8° F, the pulse rate 100 and the respiratory rate 20. Physical examination revealed an obese woman who seemed subacutely ill. There was tenderness to deep pressure over the whole abdomen with slight rigidity and moderate rebound tenderness. The white blood cell count was 12,800 per cu. mm. with 90 per cent of polymorphonuclear leukocytes. There were occasional crists in the urine.

Appendectomy and drainage were carried out under spinal analgesia through a McBurney incision 24 hours after the patient entered the hospital. The appendix was ruptured, there was a localized peritonitis and three fecal liths were free in the peritoneal cavity.

The patient was treated with measures for peritonitis including transfusions. The drainage was foul and scanty and a severe wound infection and cellulitis developed with rising temperature and increasing distention. On the eighth postoperative day multiple incisions were made in the abdominal wall.

The following day the patient developed jaundice. On the twelfth day the icteric index was 125 units and the CO combining power of the blood was 36 volumes per cent. Distention was not relieved by any of the measures employed and jaundice became deeper. Death occurred on the eighteenth day. The antemortem temperature was 107° F. The death certificate was signed out as acute yellow atrophy of the liver. Postmortem examination revealed toxic hepatitis and toxic nephrosis and a small area of localized peritonitis. The normal architecture of the liver was completely obliterated.

*Case 24.* A white man 32 years of age had suffered for 2 weeks with a dull aching pain about the umbilicus and radiating to the right lower quadrant. Twelve hours before he entered the hospital the pain became sharp. He had no other symptoms. The temperature was 98.8° F, the pulse rate 90, and the respiratory rate 24. Physical examination revealed slight rigidity, extreme tenderness, hyperesthesia and moderate rebound tenderness in the right lower quadrant. The white blood cell count was 15,200 per cu. mm. with 78 per cent polymorphonuclear leukocytes.

Appendectomy was performed through a McBurney incision under spinal analgesia. The appendix was acutely inflamed.

followed much the same anatomic path as in Jemerius case to present at the external inguinal ring but they did not have peritoneal sacs

As Carlucci and other studies indicate hernia in spite of a rather general belief to the contrary does occur after the McBurney incision. This incision was used in every instance but one of the postoperative hernias reported by McClure and Altemeier (in 46 per cent of those who survived spreading peritonitis 44 per cent who survived abscess and 18 per cent who survived local peritonitis). All of the 22 hernias following appendiceal abscess reported by Chenoweth occurred after the same incision.

Repair is carried out by the usual methods for the repair of ventral hernia. The cicatrix is excised and the layers of the wound are carefully denuded. The peritoneum should always be opened and adhesions between the parietal peritoneum and underlying viscera should be released. The hernial sac is then cut off flush with its neck and the peritoneum and other layers are sutured as in any fresh wound. It is essential that the sutures should be placed without tension and it is wise to use retention sutures to relieve the strain on the aponeurotic layer. Gallies fascia lata strips may be used in large hernias.



sions The aponeurotic layer should be overlapped by buried sutures which may be continuous in case. Deaver recommended special attention to the inner layer of the lateral portion of the rectus sheath which retracts to a considerable distance much farther than the median half of the sheath.

Abdominal wall defects are much more frequent after surgery for appendicitis than they are usually supposed to be. In a study of 700 appendectomies performed through incisions in the lower right quadrant Carlucci found that 83 patients about 12 per cent had developed postoperative abdominal wall defects ranging from simple weakness to hernias involving the whole length of the incision. The incidence was twice as great when split rectus incisions as when intermuscular incisions were used. Defects were twice as frequent in men as in women. Respiratory complications, fecal fistulas *per se*, and pregnancy and parturition did not seem to increase the incidence of hernias but many hernias followed drainage of appendiceal abscesses in which the abdominal wall was loosely sutured or not sutured at all. Weaknesses of the wall and small hernias tended to become less as time passed though sometimes on the other hand hernias developed a year or more after the operation.

Of the 83 defects reported by Carlucci only 32 were instances of true incisional hernias though the author believed that many defects classified as bulges were really hernias with large openings or instances of incomplete wound disruption in which the peritoneum and overlying muscle had given way while the external oblique fascia or the anterior sheath held secure. Thirty eight defects 16 of which were hernias occurred in 386 McBurney incisions and 33 defects 12 of which were hernias occurred in 250 right rectus incisions. Four hernias occurred in 5 transverse incisions.

A type of hernia apparently not previously recorded after appendectomy has recently been reported by Jemerin. On first inspection it seemed an ordinary right indirect reducible inguinal hernia. The patient had been submitted to appendectomy and drainage some years earlier. At operation a true incisional hernia was found which descended from the lower end of the right rectus scar behind the rectus internal oblique and transversus muscles to enter the inguinal canal and make its exit through the external inguinal ring. In contrast to the 8 post-appendectomy interstitial inguinal hernias following McBurney incisions which had been reported by Fisher Jemerin's hernia was completely enclosed in a peritoneal sac. Fisher's hernias contained omentum which

days The temperature was  $98.2^{\circ}\text{F}$  the pulse rate 80 and respiratory rate 30 Physical examination revealed deep tenderness over McBurney's point The white blood cell count was 20,450 per cu mm with 76 per cent polymorphonuclear leukocytes

The condition was considered subsiding acute appendicitis and operation was delayed for four days after the patient entered the hospital Then it was carried out under spinal analgesia through a McBurney incision The appendix was gangrenous and the distal two-thirds sloughed off as it was touched The stump was buried in dense adhesions and was apparently well walled off It was therefore not disturbed and only drainage was instituted

Two days after operation abdominal distention developed and a diagnosis of localized peritonitis was made For the next 8 days the patient's condition was quite satisfactory the temperature was below  $100^{\circ}\text{F}$  and the pulse rate below 100 On the eighth postoperative day distention increased On the twelfth day the patient had a chill and began to vomit copiously At this time the abdomen was distended and rigid but was not tender A mass was felt in the appendiceal region The white blood cell count was 20,750 per cu mm with 79 per cent polymorphonuclear leukocytes The patient was beginning to show some improvement when he suddenly collapsed and died within a few minutes The temperature antemortem was  $95^{\circ}\text{F}$  the pulse rate was uncountable and the respiratory rate was 30 The circumstances of death did not suggest embolism and the assumption was that an abscess at the site of the appendiceal stump had ruptured into the peritoneal cavity

The treatment of complicated acute appendicitis like the treatment of the uncomplicated disease is also surgical but it has ceased to be simple As has been pointed out elsewhere some surgeons some of whom are highly experienced take the position that in certain carefully selected cases a period of nonsurgical treatment is necessary to permit nature aided by replacement and other adjuvant therapy to attempt localization of the infectious process The majority of surgeons favor prompt operation regardless of the stage of the disease The arguments for and against conservative treatment have already been set forth (p. 203)

If operation is performed in advanced cases a period of rehabilitation is frequently necessary for the patients are sometimes so ill that the mere act of operation might in itself be fatal instead of lifesaving This period however should be as brief as possible and preferably should not exceed a few hours Surgery is then carried out modified to meet the conditions Whenever it can be done without undue risk the appendix should be removed The patient's state as a rule is far less jeopardous if the source of infection is removed than if the appendix is

## (VI)

### *The Therapy of Acute Appendicitis and Its Complications*

#### I SURGICAL TECHNIQUE AND ALLIED CONSIDERATIONS

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##### GENERAL PRINCIPLES

THE treatment of uncomplicated acute appendicitis needs no extended discussion. It is exclusively surgical. It consists merely of the removal of the appendix and that at the earliest possible moment even if the abdomen must be opened only on suspicion though the suspicion of course must be reasonably well founded. The principle on which the surgeon must work is that the only really safe appendix is the one which has already been removed. When this policy is followed there will inevitably be a certain proportion of unnecessary, though justified and proper operations. It is absolutely impossible in a certain proportion of cases of acute appendicitis to tell from the history, the physical findings, the laboratory data or anything else the state of the inflamed appendix at a given time or to predict what a day or even an hour may bring forth. The complications of acute appendicitis can usually be recognized by even an inexperienced surgeon. The most experienced physician cannot tell when they are going to occur.

For physicians and surgeons who accept the validity of these arguments the only conservative treatment of acute appendicitis as Finney Jr. expressed it, is radical immediate appendectomy in every case as soon as the patient is seen. There is almost no procedure in the whole range of surgery Heyd has pointed out which secures so much of present and future safety for the patient with so little loss of time and at so small a risk. The only justification for delay would be such clear cut improvement in the patient's condition that he seems well on the road to recovery although even in those circumstances as the following case shows things are not always what they seem.

CASE 25 A white man 32 years of age had been ill for 3 days with anorexia, nausea and vomiting, following the gradual onset of pain in the right lower quadrant. The physician whom he consulted on the fifth day of illness advised him to enter the hospital at once but he delayed three additional

alcohol or ether is followed by whatever antiseptic preparation the surgeon may prefer. Sterile drappings are applied according to the individual habit. The skin edges of the wound must be protected with unusual care because of the risk of contamination.

### INCISIONS

There is an increasing belief that details of surgical technique do not alter the outcome in acute appendicitis in any way whatsoever. Equally good results have been reported with all types of incisions and with wide variations in other steps of the operative procedure. None of these considerations is of importance as compared with the duration of illness before the patient is seen by the physician, whether a purgative has been taken and how promptly operation is carried out. There is no reason why the surgeon who devotes his chief attention to these basic considerations should not develop and become expert in any technique which he personally finds most satisfactory.

The criteria of any abdominal incision should be that it provide adequate exposure, conserve the strength of the abdominal wall, be fashioned as easily as possible, be closed without difficulty, permit the patient to be comfortable after operation and heal with a minimal scar. The chief objective of all appendectomy incisions is to provide access to the peritoneal cavity immediately over the inflamed appendix. This very objective should prevent the dogmatism with which types of incisions are often discussed. The wide variability of the position of the appendix and the cecum (figs 8-17 pp 21-25) under normal circumstances has already been discussed and in view of this fact it is particularly important that exposure be adequate. Out-of-sight manipulations, particularly when the entire appendix is involved, may result in the rupture of a friable and gangrenous organ into the unprotected peritoneal cavity. Operations performed early in the illness when no complications are anticipated can be carried out through a small opening if that is the surgeon's choice. Operations performed in advanced stages when difficulties are anticipated require better exposure which should be provided by a longer incision or by an incision which is readily extensible. A short incision does not avoid trauma; it is frequently a sure method of introducing it.

McBurney himself wrote in 1894:

Incisions should be long enough to allow complete and safe work to be done and it is most unscientific and harmful to encourage those of limited experience to believe that a special measure of good goes with a special

permitted to remain and to continue to pour out its infected contents into the peritoneal cavity

### ANESTHESIA

The type of anesthetic for appendectomy is determined by the preference of the surgeon with the proviso that profound anesthesia is undesirable at any time and is dangerous in debilitated patients particularly if they are advanced in years. It is also unnecessary. Perfect relaxation can be secured by a combination of pre-operative sedation and the use of one of the gaseous anesthetics such as nitrous oxide ethylene or cyclopropane with all of which varying quantities of oxygen are administered and all of which can be supplemented with appropriate amounts of ether.

Spinal analgesia may be used if that is the surgeon's preference but the contra indications should be carefully borne in mind. This type of analgesia is sometimes advocated for complicated acute appendicitis on the ground that the intestines are kept in a state of contraction and therefore do not crowd into the incision. That is quite true but in view of the fall in blood pressure which may accompany even carefully supervised spinal analgesia the method is not without risk in patients whose condition is poor. It must be remembered too that this type of analgesia is not *per se* a protection against pulmonary complications actually the diminution of the diaphragmatic excursions which are associated with its use may predispose toward the development of such complications.

A surgeon accustomed to local analgesia may prefer to use it in uncomplicated cases of acute appendicitis. In complicated cases the choice would be open to question. Relaxation with this method is not satisfactory and it predisposes to the spread of infection. Neither this method nor regional block analgesia should be attempted by surgeons who are inexperienced in their use.

### LOCAL PREPARATION

Local preparation of the operative field is the same as for any other abdominal operation except as it is modified by the emergency character of the procedure. The skin is shaved over the entire abdomen including the pubic area in the event that an incision outside of the lower right quadrant may be necessary because of a mistaken diagnosis. Cleansing with soap and water the excess of which is removed by

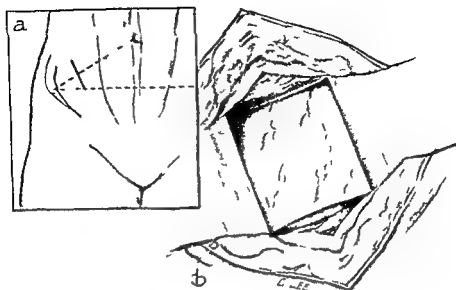


Fig 58 McBurney incision for appendectomy showing a location of incision and b retraction of internal oblique and transversalis muscles preliminary to incision of peritoneum

inexperienced is the surgeon the more important are these considerations

Whenever a muscle splitting incision is used regardless of the location scrupulous attention must be paid to the protection of the nerve supply Injury to the ilio inguinal and iliohypogastric nerves not only causes postoperative discomfort and pain but may also lead to diffuse weakness of the entire right lower quadrant of the abdominal wall and to subsequent herniation No single kind of incision incidentally protects against hernia or disruption of the wound (p 255)

*McBurney (McArthur McBurney) Incision* The first operations for appendicitis were usually carried out through incisions parallel and medial to the lateral edge of the right rectus muscle McBurney observed that with this incision the operator was forced to work beneath an overhanging shelf formed by the outer part of the abdominal wall since the appendix usually lies to the right of the incision often close to the outer part of Poupart's ligament or pointing upward to the outer side of the colon He therefore devised the incision which bears his name (fig 58) though since it was also devised independently a few

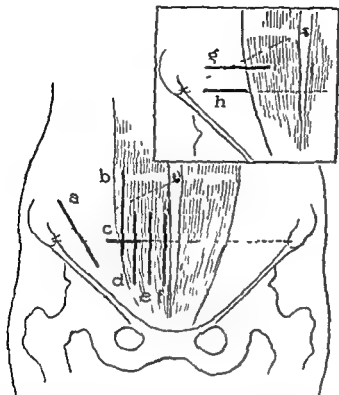


Fig 57 Incisions for appendectomy a McBurney b Murphy c Davis d Battle e right rectus f paramedian g Rocky h Elliott

length of incision If the parts severed in the making of a wound are properly adjusted and the wound properly treated repair will be just as rapid and complete whether the wound be five inches long or three inches long while on the other hand no good surgeon will ever unnecessarily divide tissue simply because he can get an obtuse repair

The appendix can be reached through almost any incision made in the right lower quadrant of the abdomen (fig 57) within an area bounded by lines drawn (1) from the umbilicus to the symphysis pubis (2) from the symphysis along Poupart's ligament to the anterior superior spine of the ilium and (3) from the umbilicus round to a point on the crest of the ilium midway between the anterior and posterior iliac spines (Strohl) The incisions themselves may be of three types (1) oblique muscle splitting (2) through the outer fibers of the right rectus muscle or through its sheath with retraction of the muscle medially and (3) transverse

The reason for employing any incision other than that of McBurney in the performance of an appendectomy is to secure better exposure and to avoid the embarrassment and actual risk of trying to remove an adherent displaced appendix through an unsuitable incision The more

and various methods of extending it have been devised (chiefly the methods of Weil and Harrington) but the technique described by McBurney is still the basic procedure for all incisions of this kind

In view of some of the claims made for the McBurney incision it is interesting to observe that as the author described it it is anything but a buttonhole incision and that he himself stated This operation does not appear to be suitable for cases accompanied by suppuration about the appendix which require to be treated by extensive picking with gauze nor in cases nonsuppurative which require during operation a large intra abdominal dissection Its advantages as he listed them are as follows

In the first place muscular and tendinous fibers are separated but not divided so that muscular action cannot tend to draw the edges of the wound apart but rather to actively approximate them Excepting during the incision of the skin almost no bleeding occurs The fascia transversalis not being drawn away by the retraction of the deepest layer of muscular fibers the fascia is easily completely sutured and thus greater strength of repair is assured No muscular fibers or larger nerves having been divided pain after operation is almost absent

The McBurney incision is entirely satisfactory for an appendix located in the so called normal position in the right iliac fossa but whether or not one comes down at once on the appendix seems often to be as much a matter of good luck as of surgical judgment Mason and his associates who use this incision routinely had to enlarge the original wound only 16 times in 545 cases Others have been less fortunate

This is a difficult incision to work through particularly if the surgeon is inexperienced when the appendix is retrocecal when it is deep in the pelvis or when it is located high under the liver and the difficulties of removal are increased if the patient is obese The subhepatic location of the appendix may be distinctly unusual but I recollect with embarrassment that twice within a single school term when operating before students I was obliged to close a McBurney incision and make a right rectus incision because the appendix was densely adherent to the under surface of the liver The removal of an adherent gangrenous pelvic appendix through a McBurney incision is also attended with special difficulties and risks When on the other hand the appendix is readily accessible the McBurney incision has definite advantages The cecum is approached from its outer aspect so that manipulations of the small bowel are reduced to a minimum and the chances of spreading the infection and of postoperative ileus are correspondingly



months earlier by McArthur (p 15) it should properly be called the McArthur McBurney incision. McBurney described it as follows:

The incision in the skin is an oblique one about four inches long. It crosses a line drawn from the anterior iliac spine to the umbilicus nearly at right angles about one inch from the iliac spine and is so situated that its upper third lies above that line.

The section of the external oblique muscle and aponeurosis should correspond great care being taken to separate these tissues in the same line *not cutting any fibers across*. This is easily accomplished.

When the edges of the wound in the external oblique are now strongly pulled apart with retractors a considerable exposure of the internal oblique muscle is seen; the fibers of which cross somewhat obliquely the opening formed by these retractors. With a blunt instrument such as the handle of a knife or closed scissors the fibers of the internal oblique and transversalis muscles can now be *separated* without cutting more than an occasional fiber in a line parallel with their course—that is nearly at right angles to the incision in the external oblique aponeurosis. Blunt retractors should now be introduced into this in turn and the edges separated.

The transversalis fascia is thus well exposed and is then divided in the same line. Last of all the section of the peritoneum is made.

Two sets of retractors must be in use: one holding open the superficial wound from side to side; the other separating the edges of the deeper wound from above downward. A considerable opening is thus formed through which in suitable cases the cecum can be easily handled and the appendix removed. The appendix having been taken away the wound in the peritoneum which is transverse is then closed by suture. The similar wound in the fascia transversalis is also sutured. The fibers of the internal oblique and transversalis muscles fall together as soon as the retractors are withdrawn and with a couple of fine catgut stitches the closure can be made more complete. The wound in the external oblique aponeurosis is sewed with catgut from end to end. When the operation is completed it will be seen that the gridiron-like arrangement of the muscular and tendinous fibers to which the abdominal wall largely owes its strength is restored almost as completely as if no operation had been done.

The opening into the peritoneal cavity is not large but may be made larger if necessary by continuing the separation of the fibers of the internal oblique and transversalis and dividing the conjoined aponeurosis in the same line with scissors. In the opposite direction the separation of muscular fibers may be carried out as far as the crest of the ilium.

Most of the oblique muscle splitting incisions introduced since McBurney's day are merely modifications of the incision which he and McArthur described. It has been recommended that the incision be made higher in the abdomen or closer to the anterior superior spine.

rectus sheath but slightly nearer to the median line (5) The abdomen is closed in layers The peritoneum and subperitoneal connective tissue are closed en masse Each layer of the rectus sheath is sutured separately the site of the incision in the posterior sheath being protected by the rectus muscle as it slides back into place The skin and subcutaneous tissue are sutured separately

The nerves encountered in the course of the incision which usually are two branches of the twelfth intercostal nerve or branches of the eleventh and twelfth intercostal nerve are pushed out of the way Great care must be exercised not to cut them The deep epigastric vessels which lie upon the posterior rectus sheath are also removed from the operative field by being drawn inward along with the rectus muscle

*Right Rectus Incision* The right rectus incision differs from the Battle incision in only three respects (1) The incision is made nearer the center of the rectus muscle (2) The muscle is divided in the direction of its fibers (3) The fibers of the muscle are retracted medially and laterally The chief difference in other words is that in the Battle incision the whole muscle is retracted medially whereas in the right rectus incision the fibers are split and are retracted medially and laterally Murphy beginning with his first operation in March 1889 routinely used an incision through the outer margin of the rectus one half inch to the central side of the linea semilunaris and Lennander and Edebohl's also preferred to divide and separate the fibers of the muscle in line with the skin incision

*Paramedian Incision* In this incision the skin subcutaneous tissue and anterior rectus sheath are divided  $\frac{1}{2}$  to  $\frac{3}{4}$  inch from the medial border of the right rectus muscle The anterior rectus sheath on the medial aspect of the incision is separated from the underlying muscle until the free edge of the latter appears The medial aspect of the rectus muscle is then freed from its bed for a short distance and is drawn outward by a retractor The exposed posterior rectus sheath and the peritoneal layer are divided en masse in a line corresponding with the incision of the anterior rectus sheath The abdomen is closed in layers by the usual method the muscle being replaced in its former bed over the suture line in the peritoneum and posterior rectus sheath

*Transverse Incisions* The first transverse incision for appendectomy which was described by Elliot in 1896 is carried out as follows A horizontal incision is made through the skin and the aponeurosis of the external oblique muscle beginning  $\frac{1}{2}$  inch inside the anterior superior spine of the ilium and extending to the linea semilunaris The apo

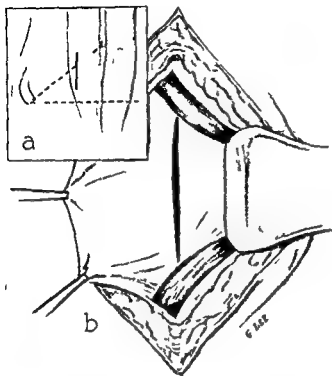


Fig 59 Battle incision for appendectomy a location of incision b medial retraction of rectus muscle preliminary to incision of posterior rectus sheath and peritoneum

reduced. If adhesions have formed the appendix can be removed without disturbing them and since the abdominal contents lie medial to the incision the area of operation (and infection) can be fully walled off. The layers fall promptly into place and closure is correspondingly simple. The chances of hernia are probably less than with other varieties of incision even if drainage is used though as already noted they do occur.

**Battle Incision.** In 1895 Battle described a longitudinal incision for appendectomy consisting of the following steps (fig 59) (1) A longitudinal incision is made  $1\frac{1}{2}$  inches to the inner side of the linea semilunaris. The center of the incision corresponds to a transverse line drawn between the anterior superior iliac spines. The skin, subcutaneous tissue and superficial fascia are opened en masse. (2) The anterior sheath of the rectus muscle is divided parallel to the skin incision. (3) The rectus muscle is retracted medially exposing the posterior layer of the rectus sheath and the transversalis fascia. (4) The posterior rectus sheath, the subserous connective tissue and the peritoneum are opened en masse parallel to the incisions in the skin and the anterior

**linea semilunaris** The aponeurosis of the external oblique muscle is divided in a line with the skin incision but obliquely to the direction of the fibers. The fibers of the internal oblique and transversalis muscles are parted (not cut) in the same line as the overlying structures. The incision is carried inward through the anterior layer of the rectus sheath. A blunt retractor is inserted and the muscle is drawn toward the median line exposing the posterior rectus sheath which is divided with the peritoneum. A triangular opening is thus provided with a base measuring  $\frac{3}{4}$  inch and sides measuring 1 inch each. For more difficult cases an incision 4 to 5 inches long can be made by prolonging the outer edge of the incision to the anterior iliac spine or higher and inwardly through the sheath of the rectus muscle to within an inch of the median line.

The Rockey incision begins with a transverse incision carried through the skin and deep fascia directly across McBurney's point with the inner end over the belly of the rectus and the outer just above the iliac spine the length varying with the requirements of the special case. The only structures cut are the rectus sheath the aponeurotic junction at the outer border and the aponeuroses of the muscles. The incision is then spread wide open by an up and down pull just as one would open the drawstring of a purse so that the muscle fibers are separated and the peritoneum is exposed. The Rockey technique is thus substantially the same as the Davis technique the chief difference being in the location of the incision.

Both authors claimed important advantages for this type of incision including a minimum loss of blood the speed with which the operation can be performed the protection of nerves blood vessels and other structures the ease of access and the absence of the intestines from the operative field. Both also claimed in effect that whatever the position of the appendix the center of the incision crosses McBurney's point which is the center of surgical action. On the other hand Rockey admitted that this type of incision is not suitable for the removal of a pelvic appendix and many would take issue with his unqualified statement that it can be determined before operation that the appendix occupies this (or any other) position. Rockey also stated that if operation has been performed on an indeterminate diagnosis a small scar is a strong and well healed abdominal wall is not a reproach to the surgeon's conscience or a reminder of his uncertainty of diagnosis. This is no particular recommendation to a surgeon who has diagnosed the case to the best of his ability and then performed the operation with the secure knowledge that he was acting in the patient's best interests.

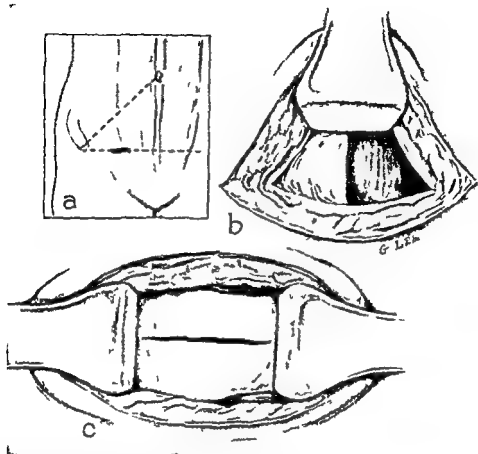


Fig 60 Davis incision for appendectomy a location of incision b retraction of internal oblique and transversalis muscles preliminary to incision of peritoneum c location of peritoneal incision

neurosis of the external oblique is divided and the fibers of the internal oblique and the transversalis muscles are separated as in the usual McBurney incision. The peritoneum may be opened by either a transverse or a longitudinal incision.

Much the same situation exists concerning the transverse incision for appendectomy introduced by Davis and Rockey, which has already been described for the McArthur McBurney incision (p 15). The description of Rockey's incision was first published in November 1905 and the description of the similar incision devised independently by Davis was published in January 1906.

The Davis incision (fig 60) begins with a transverse incision 1½ inches long through the skin and subcutaneous tissues on a level with the anterior superior spine of the ilium with the center on the right

should then be followed which may include any or all of the following methods as necessary

1 The first part of the ascending colon to be encountered is drawn out of the wound and is followed down to the cecum. The anterior of the three longitudinal bands of the colon (*taeniae longitudinales*) will lead directly to the base of the appendix which is 2 to 3 cm. below this point. When once these bands are located there is seldom further difficulty in identifying the appendix. The fold which runs from the terminal ileum to the cecum (p. 29) may also serve as a landmark.

2 The index finger is inserted and directed outward so that it passes along the anterior lateral and posterior parietal peritoneum until the cecum is found and lifted into the wound.

3 The fingers follow the parietal peritoneum posteriorly beginning opposite the anterior superior spine of the ilium and working toward the midline. The fold of peritoneum holding the cecum (not the meso-appendix) is usually thus located and if the fingers are hooked under it and it is pulled gently upward and into the wound the appendix is frequently brought into view with it.

4 If the bowel which first presents in the wound is believed to be the sigmoid colon or the transverse colon it can be recognized by its free mesentery. If the bowel is the sigmoid colon it becomes taut in the direction of the pelvis as delivery is attempted.

If there is great difficulty in identifying the presenting portion time is saved by using Monks' method. The surgeon draws out of the abdomen the coil under investigation holding the ends between the fingers of the right and left hand. At the same time an assistant passes his hand down along the mesentery to the spine to be sure that there is no twist. If there is none the proximal or upper end of the coil will be in the surgeon's left hand and the distal or lower end in the right and the investigation can be continued from those landmarks.

5 Payne after finding them in 1000 successive laparotomies pointed out that in the terminal portion of the ileum there will be found from one to three small straight vessels springing from the anastomosis formed by the ileocolic and right colic arteries. From the pylorus downward in the small bowel to a point about 14 inches from the ileocecal valve the visible vessels run at right angles to the long axis of the bowel. In contrast the vessels which Payne described run parallel to the long axis of the bowel immediately beneath the peritoneal coat. They are definite end arteries and their distinctive location and course make them useful in locating the appendix more useful in fact than

## REMOVAL OF THE APPENDIX

*Identification of the Appendix* As soon as the peritoneum has been opened the wound is separated by retractors to permit easy inspection of the structures. If the appendix comes immediately into view the finger can be inserted under it or it can be gently lifted to the surface for closer inspection. Fresh plastic adhesions holding a visible appendix *in situ* may be separated by blunt dissection and ligated if necessary. Bands are doubly clamped, divided and then ligated. If however the appendix is not in full view these maneuvers should not be attempted for a friable gangrenous appendix ruptures readily and an appendix distended with pus especially if the pathologic process has reached the outer coat may also rupture and its contents be spilled into the unprepared peritoneal cavity if out of sight manipulations are attempted.

If the cecum presents in the wound and is recognized as such it may be lifted out with the fingers or with rubber covered intestinal forceps. A search is then made for the appendix; it is simplified if the intestine is put on stretch. The appendix is first sought behind the cecum or the terminal ileum. If it is not in these locations it is looked for in the pelvis or under the liver. If the cecum lies high its lower pole may be above the level of the umbilicus and the appendix is likely to be found in the ascending or subhepatic position. If other methods fail the lateral parietal peritoneum may be incised and the cecum freed and rolled inward and upward when this maneuver is carried out the appendix is often found in the retroperitoneal connective tissue or in the posterior cecal wall.

In the search for the appendix a finger can be inserted into the incision and carried down as far as possible along the anterior parietal peritoneum and then back upward along the pelvic brim until the iliac vessels are recognized by their pulsation. The finger then continues upward until the ileocolic mesentery is encountered. This mesentery is followed outward toward the ileocecal junction. By another maneuver the fingers are passed along the parietal peritoneum from the outer to the inner side of the wound across the iliac fossa. Here the cecum can be located and after it is drawn into the wound the appendix is identified by tracing the longitudinal bands downward to its point of junction with the cecum.

Sometimes the bowel which first presents in the wound is not the cecum and considerable difficulty may be experienced not only in locating the appendix but in identifying the cecum. A systematic plan

pendix with abscess formation. The patient died 7 hours after operation. Post mortem examination confirmed the diagnosis and revealed also a localized fibrinopurulent peritonitis.

*Isolation of the Appendiceal Region* As soon as the structures are identified the cecum is held out of the way so that it will not be packed away with the small intestine and a long sponge forceps is introduced by means of which a moist gauze roll is carried toward the ileum and along the posterior abdominal wall toward the midline. Free loops of small bowel are pushed ahead of the pack. Enough gauze is introduced to permit the erection of a protective wall on the inner aspect of the appendiceal region so that the general peritoneal cavity and loops of small intestine are completely blocked off. A second similar barrier may be built up toward the liver fitting against the first wall and extending out into the flank. A third wall blocks off the true pelvis. Packing should not be omitted in any case. It is clearly necessary in the contaminated case but it may prove equally necessary in the supposedly clean case in which accidental rupture occurs or untoward bleeding complicates the procedure.

*Ligation and Division of the Meso appendix (fig 61)* As soon as the appendix has been delivered outside of the peritoneal cavity a clamp is applied to the free edge of the mesentery. A clamp should never be applied to the tip of the appendix especially if the organ is distended or gangrenous.

The mesentery is ligated en masse or in sections according to its size and vascularity. If it is unusually short it must be ligated and divided within the abdomen before the appendix can be delivered. If a clamp is applied distal to the point of ligation and division is carried out between the clamp and ligature back bleeding which is sometimes very troublesome is completely eliminated. Ligatures must be applied with special care if the meso appendix is friable and it is usually wise to place an additional interrupted suture at the mesenteric border close to the base of the appendix to secure the intramural branch of the posterior cecal or appendicular artery.

After the ligatures are placed the appendix is dissected free from the mesentery preferably from the tip to the base. The dissection is carried 1 cm or more beyond the ceco appendiceal junction to allow room for the placing of the pursestring suture in the cecum. If the segment of the appendix near the base has no mesentery but is attached to the cecum by a common peritoneal covering the dissection must be carried out until the point of entrance into the cecum is clear. The raw



following the longitudinal muscle bands of the large bowel since the last 1½ inches of the terminal ileum furnish the most direct guide to the base of the appendix

6 A simple method of identifying the appendix if it is not readily found is to pick all the movable abdominal contents toward the patient's left side. The cecum which is more or less fixed in the right iliac fossa remains immobile and is finally identified by elimination of the other movable contents. This procedure according to Ashhurst (cited by Royster) is the least brilliant but the surest method of identifying the cecum and appendix.

Every effort should be made to handle the intestines as little as possible and it is always desirable when circumstances permit that only the cecum and terminal ileum be seen within the wound. Some surgeons believe that this is more readily achieved if operation is performed in the Trendelenburg or even in the semilateral position. Gravity then assists in keeping the small intestine out of the operative field and an infectious exudate is kept out of clean parts of the cavity. On the other hand regardless of the difficulties of the search the appendix must be sought for until it is identified in any instance in which the diagnosis of acute appendicitis has been introduced. In 3 cases in the Charity Hospital series ileostomy for supposed intestinal obstruction was done without a search for the appendix and the rupture of the appendix which was responsible for the intestinal distention was not found until autopsy. The following case is another in which operation was done on a diagnosis of intestinal obstruction.

**Case 26** A white man 18 years of age had been ill for 7 days with abdominal pain, nausea, vomiting, diarrhea and abdominal distention. The pain had begun in the epigastrium and 24 hours later localized in the right lower quadrant. The diarrhea was checked by paregoric after it had been present for 3 days. The patient had lost 16 pounds since the beginning of his illness. The temperature was 98.6° F, the pulse rate 82 and the respiratory rate 20. The tonsils were enlarged and cryptic. The abdomen was distended, tympanic and generally tender, the tenderness being most marked in the right lower quadrant and across the pelvis.

Roentgenologic examination of the abdomen disclosed marked distention of the small intestines and multiple fluid levels and a diagnosis of partial intestinal obstruction was made. Intestinal decompression and other conservative methods were instituted with some improvement. On the fifth day of hospitalization the patient took a marked turn for the worse and the vomitus assumed a fecal character. On the seventh day ileostomy was carried out under ether anesthesia through a right rectus incision. The obstruction was located in the terminal ileum and originated in a gangrenous and ruptured ap-

method it should be remembered that the appendiceal blood vessels are located medially to the organ and dorsal to the cecum and colon and their blood supply. Some surgeons prefer to apply the pursestring and amputate the appendix before performing retrograde separation, but the risk of contamination is greater when this plan is employed.

McNealy and Lichtenstein called attention to the possibility of producing angulation of the cecum when the mesentery of the appendix is ligated, particularly if the meso-appendix is short or if the ileo-mesenteric fold is clamped and ligated with the meso-appendix. They have observed acute mechanical obstruction when this has been done. To prevent the accident they recommended that the ligature on the meso-appendix should not include the ileo-appendiceal or ileomesenteric fold. They also recommended that if the meso-appendix is broad the vessels should be ligated separately, since en masse ligation may include the ileal attachment and disturb the normal relationship between the ileum and cecum.

#### MANAGEMENT OF THE APPENDICEAL STUMP

Variations in the technique of appendectomy are chiefly concerned with the management of the appendiceal stump. Three chief methods are in use: simple ligation, ligation and inversion, and inversion without ligation. The dozens of different methods which have been described differ only in details from these three basic methods, and many of the modifications suggested make the operation unnecessarily complicated.

In 1908 Willis, in response to a questionnaire, found that 73.3 per cent of the 105 surgeons who replied always buried the stump and 10.3 per cent usually buried it. The ratios today are probably about the same.

By the method most often used, a pursestring suture of linen or silk is inserted from  $\frac{1}{8}$  to  $\frac{1}{2}$  inch from the base of the appendix through the seromuscular coat of the cecum, including the submucosa, and catching each longitudinal band (fig. 62). The base of the appendix is gently crushed between the jaws of a clamp and a ligature of plain O catgut is placed about the crushed area. Two additional clamps are applied above the ligature. The appendix is divided between them with a knife, after which the stump is touched with an applicator dipped in pure carbolic acid, followed by alcohol on an applicator. If an inflammable anesthetic is not being used, the appendix may be amputated with the cautery (fig. 62C).

After the stump of the appendix has been inverted into the cecal

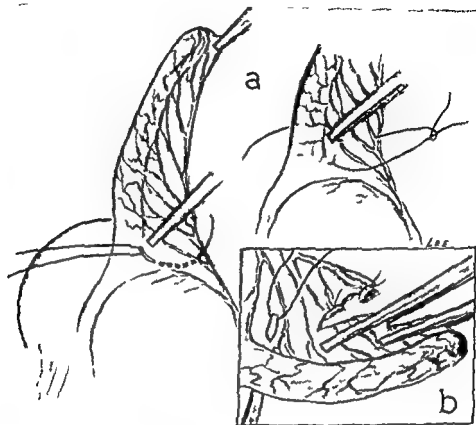


Fig 61 Technique of appendectomy a en masse ligation of meso appendix b segmental ligation of meso appendix

surface on the cecum is covered over with Lembert sutures so placed as to guard against producing a stenosis of the ileocecal orifice. Otherwise the stump of the mesentery is later sutured over the site at which the appendiceal stump is inverted.

If the base of the appendix presents in the incision while the remainder of the organ is bound down in the pelvic region or retrocecal fossa the usual tip to base method of removal may not be practical and retrograde removal must be carried out. The base of the appendix is cautiously freed by blunt dissection at the ceco-appendiceal junction after which a probe is passed through the meso-appendix, a clamp is applied to the base of the appendix and the meso-appendix is ligated en masse or segmentally. The remainder of the appendix is then separated from the meso-appendix by carrying out the usual technique in reverse. When a retrocecal appendix is removed by the retrograde

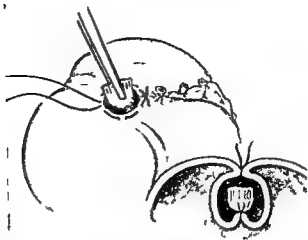


Fig 63 Technique of appendectomy. Inversion of appendiceal stump into cecum

not occur and convalescence should be smooth adhesions are likely to form about the exposed stump and may give rise to later intestinal obstruction

When inversion with or without ligation is practiced an infected structure (the stump) is placed in the cecal wall (an enclosed cavity) and several undesirable consequences may follow. The principles of intestinal surgery again are violated in that mucosa is permitted to come into contact with serosa. If the appendiceal stump has been cauterized the placing of devitalized tissue in an enclosed space may be followed by anaerobic infection. An inflammatory reaction about the stump may extend to the cecum and an intramural abscess may form. Robertson (cited by Mayo) who studied the appendiceal stump at postmortem examination when death had occurred from causes unconnected with appendectomy found pus in the pocket in the cecal wall as long as 21 days after operation. Mayo who recommended the inversion technique also called attention to the fact that the suture used to invert the stump having passed through the intestinal wall is necessarily contaminated.

All of these objections are apparently valid but most of them seem theoretic in view of the hundreds and thousands of cases in which simple ligation and more particularly ligation and inversion are practiced with only a minimum of accidents. It would seem reasonable to conclude that if whatever technique is chosen is carried out carefully and with due regard to surgical principles the end results are likely to be the same.

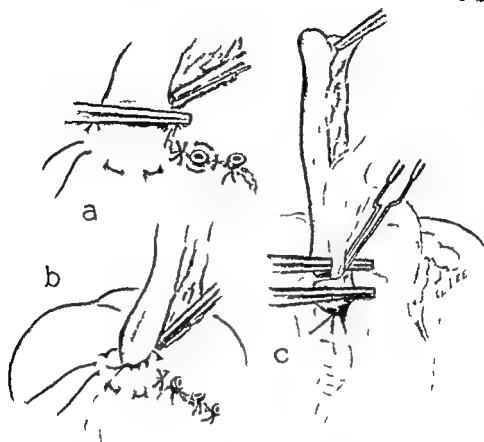


Fig 62 Technique of appendectomy a crushing of base of appendix after application of pursestring b ligation of base of appendix c cauterized division of base of appendix between clamps

wall with a smooth tissue forceps (fig 63) the ends of the pursestring suture are pulled taut and tied. The area of inversion is covered over with a second pursestring suture or with Lembert sutures as the surgeon may prefer.

It must be granted that every one of the methods used in handling the appendiceal stump is open to at least theoretic objections. Simple ligation permits a contaminated raw surface to remain free in the peritoneal cavity and also violates the fundamental principle of intestinal surgery that like surfaces should be approximated. If the ligature becomes loose or blows off or if the stump undergoes necrosis at the best a fecal fistula will occur and at the worst fatal peritonitis. Even if this accident should

## CLOSURE OF THE WOUND

In uncontaminated cases the wound is closed without drainage in layers with such modifications as may be necessary to meet the conditions introduced by the type of incision used. Whatever suture material the surgeon has found satisfactory may be used. Nonabsorbable sutures of cotton or silk must be used if early ambulation is to be practiced. Retention sutures are advisable in obese subjects or when drainage is employed.

## THE SURGERY OF COMPLICATED ACUTE APPENDICITIS

Removal of the appendix as pointed out is as desirable in advanced acute appendicitis as in the early stages of the disease. The technique described is followed as closely as possible but must be modified to meet the exigencies of the individual case. It is impossible to lay down general rules other than the warning that all manipulations should be gentle to avoid breaking down protective adhesions which may have formed.

In the ordinary case of unruptured acute appendicitis drainage is unnecessary. A clear and odorless exudate or an exudate containing flecks of fibrin is protective (p. 97) and should be left *in situ*. In fact almost the only conceivable indication for drainage in such cases would be persistent oozing at operation or uncertainty as to hemostasis.

In 1905 Yates (cited by Riggs) showed in an elaborate study that it is impossible to drain the peritoneal cavity completely by any method. Since that demonstration the discussion in regard to the necessity for and the value of drainage has continued without final settlement. With in the last decade there has been an increasing tendency to dispense with it altogether regardless of the stage of the disease or the state of the peritoneal cavity.

Drainage has certain disadvantages in addition to its proved inadequacy. Hernias for obvious reasons are more frequent when it is employed. Drains even when they are properly placed have a tendency to cause adhesions and thus may be responsible later for intestinal obstruction. If they are badly placed they may cause intestinal necrosis. Hospitalization is always prolonged when they are used. Some of these arguments are valid others are not. Considerations of convenience and expense do not enter the question when one is dealing with a patient whose life in the opinion of a competent surgeon may depend upon whether or not his wound is drained.

barring the occasional accident which may occur at any time and by any method Willis reached that conclusion as the result of his study in 1908.

The technique devised by Ochsner and Lilly is presented as typical of many other departures from the basic technique. By their method the pursestring suture is begun on one side of the meso appendix, passes down to and includes the submucosa and emerges from the opposite end of the meso appendix. The same suture is reinserted in the same manner so that it forms a loop around the mesenteric portion of the ceco-appendiceal wall and encircles any intramural branch of the appendicular artery. The pursestring is then continued around the cecum in the conventional manner.

Three clamps are applied at the base of the appendix between the upper and middle of which the organ is amputated. The crushed end of the stump is grasped by smooth tissue forceps after the second (middle) clamp is removed. The lower clamp is then removed and the crushed stump is inverted into the lumen of the cecum following which the pursestring is tightened to cover it. One or two Lembert sutures are used to invert further the already inverted stump.

The advantages of this method according to the authors are as follows. The stump is inverted into the cecal lumen without danger of infection from pocketing of the contaminated stump. Serosa is brought into apposition with serosa insuring firm healing of the cecal wound and obviating the danger of subsequent leakage from the cecum and the risk of peritoneal contamination. Contamination from the presence of an infected stump lying free in the peritoneal cavity is avoided. Careful peritonealization is secured. The infected peritoneal stump is not buried in a closed cavity. The risk of hemorrhage is obviated by the introduction of the pursestring suture in such a way as to include any intramural branch of the appendiceal artery. Finally, the risk of spillage of intestinal contents during inversion is obviated by the use of three crushing clamps with division of the appendix between the upper and middle instruments and grasping the crushed end of the stump with inverting forceps before the lower clamp is removed.

If the inflammatory process has extended to the cecum so that the wall is stiff, distended, thinned out, friable, edematous or actually gangrenous, it is difficult to introduce a nonpenetrating stitch or even to introduce any stitch at all, and the chances of a blow out of the suture cannot be taken. Either the stump must be ligated without inversion and drainage instituted or if that seems risky the operation of appendicostomy must be performed.

purpose and the extent of the pathologic process. A localized collection of pus or an oozing area needs only localized drainage. If the process is generalized, three drains are usually necessary: one to the appendix stump, another to the depth of the pelvis, and a third to the lateral colic gutter. Drainage of the retrocecal space and to the pelvis does not always prevent residual abscesses, but the general opinion is that its use is a precaution against their development. Aspiration of purulent exudates should always be carried out before drains are inserted.

As far as possible, drainage tubes should lie along the parietal peritoneal wall and should not cross or lie among loops of intestine. In the pelvis they should be placed along the posterior lateral wall. When so placed, they do not disturb protective adhesions, do not cross uncontaminated areas of peritoneum, and are not likely to cause intestinal obstruction.

Drains may be brought out through the center or the lower end of the incision, or through an additional stab wound made to the outer side of the incision in the iliac fossa, just above the pubes, or in the right flank, depending upon the indications in the individual case. If a stab wound is made in the flank to drain the retrocecal pouch, it should not be made too near the crest of the ilium, which is a highly vascular area.

*Removal of Drains.* Unless drainage has been used because oozing was free or some doubt was felt about hemostasis, it may fairly be said that drains which can be removed with safety a few hours after operation were probably never needed. If drainage has ceased by the third postoperative day and the patient's condition is good, the drainage tube may be slightly loosened and gradually removed over the course of the next few days, a small portion being cut off each day. If drainage continues free, the tube should not be loosened for at least 5 days. The amount and kind of discharge and the patient's general condition are better guides as to when drains should be removed than the time which has elapsed since operation. If they are removed too soon, tenderness, localized pain, and fever are likely to continue or to recur, indicating either the continuation of the original infection or the formation of additional abscesses. A slight elevation of temperature is to be expected when the drains are first disturbed. If multiple drainage has been employed, the drain to the lateral colic gutter is removed first, the one to the base of the appendix next, and the one to the cul de sac last of all.

Postural (prone, lateroprone) drainage of the peritoneal cavity, although never widely popular, is a rational method. It is based on the practical consideration that drainage as usually carried out violates all principles of gravity. In the prone position, as Riggs pointed out, the



Statistics as to the value of drainage or the risk of omitting it are entirely inconclusive and not worth citing

Generally speaking drainage is frequently or always indicated

(1) Whenever there is a localized abscess or a localized collection of pus

(2) When diffuse peritonitis or pelvic peritonitis with a purulent or mucopurulent exudate is present

(3) When there has been gross soiling of the peritoneal cavity by fecal material

(4) When the meso appendix or other tissues are of doubtful viability

(5) When the retroperitoneal tissue is contaminated or infected or when it has been traumatized in the difficult removal of a retrocecal appendix

(6) When there is extensive fat formation in the meso appendix

(7) When oozing has been free or hemostasis is doubtful

(8) When inversion of the stump is impossible and the development of a fecal fistula is feared

(9) Whenever for any reason an undue amount of suture material has been used

Drainage is not usually required

(1) When the appendix has been completely removed

(2) When hemostasis is satisfactory

(3) When soiling of the cavity has not occurred

(4) When the appendiceal stump has been adequately eared for

(5) When localized collections of pus are not present

(6) When a purulent exudate is not present

(7) When the peritoneal exudate is clear or turbid or when a fibroplastic peritonitis is present

The institution of drainage in relation to the bacteriologic findings in the peritoneal exudate is discussed elsewhere (p. 70)

**Drainage Material** Drains should always be of soft material such as rubber dam or soft rubber tubing and preferably fenestrated Penrose drains which consist of rubber dam wrapped about gauze are very satisfactory Tubes should be about 25 cm long and at least 1 cm in diameter Hard rubber tubing glass and similar materials should not be used Gauze should not be used unless the purpose is to create a drainage tract to the outside in such cases rubber dam is equally satisfactory and probably is capable of less harm Dressings should be loose and fluffy to permit the free exit of secretions

**Placing of Drains** Where drains should be placed depends upon their

likely to occur. As a rule the focus of infection is under the aponeurosis and within the fibers of the internal oblique muscle not in the peritoneal layer since the resistance of the tissues of the wall is less than that of the peritoneum. It is quite possible in the absence of drainage for a subaponeurotic abscess to rupture into the peritoneal cavity.

**Case 27.** A white boy 16 years of age had suffered from abdominal pain for 5 days and had been confined to bed for 2 days. He had had many similar attacks. The temperature was 99° F, the pulse rate 90 and the respiratory rate 22. Physical examination revealed deep tenderness and rigidity over McBurney's point. Rectal examination revealed bilateral tenderness. The white blood cell count was 15,500 with 80 per cent polymorphonuclear leukocytes.

Appendectomy was performed 48 hours after hospitalization under spinal analgesia through a McBurney incision. The appendix was acutely inflamed and injected. Forty-eight hours after operation in the course of a smooth convalescence the patient's temperature suddenly rose to 104.6° F. A diagnosis of atelectasis was tentatively suggested. On the fifth day vomiting and diarrhea developed. Tests for malaria and typhoid fever were negative. On the seventh day there was tenderness over the entire abdomen and other signs of peritonitis. The patient was irrational. Roentgenologic examination of the chest was negative. Examination of the wound revealed a severe infection; a wound abscess had apparently ruptured into the peritoneal cavity. The blood culture was positive for streptococci. The patient did not respond to the routine treatment for peritonitis including transfusions and the use of antistreptococcic serum and death occurred on the eleventh postoperative day.

Postmortem examination revealed generalized peritonitis, wound infection, necrosis of the wound edges, partial evisceration, purulent myositis draining into the peritoneal cavity and hemolytic streptococcic septicemia.

When the wound is to be drained the peritoneal layer is closed by the usual technique. A cigarette drain or rubber dam is then placed in the wound and the muscular and aponeurotic layers are closed loosely about it with interrupted sutures. The drain is gradually shortened and is removed when drainage has ceased. More complicated methods have been devised but are no better.

Delayed closure of the wound is frequently an effective substitute for drainage of the abdominal wall. The peritoneum and fascia are closed as usual. Deep sutures are inserted through the skin down to and through the fascia but are left untied. The wound is packed with gauze which is removed 24 to 48 hours after operation. The previously placed sutures are then drawn together and the subcutaneous tissues and skin are thus approximated.

viscera hang from their attachments in orderly folds or loops the circulation is unimpeded by the weight of the contents of the bowel and free drainage is favored from areas through which the toxic substances to be drained are most readily absorbed. When the method is used the usual drainage material is employed in the usual locations but only the peritoneal layer of the wound is closed. The patient is placed in the prone position for 12 hours alternately in the prone and right lateral position for the next 36 hours and in these positions at intervals for the next 24 to 72 hours. Several series of cases in which the prone position was used showed considerable improvement in mortality in the period when the general mortality of peritonitis was disturbingly high. The necessity for the position which at best is inconvenient and uncomfortable has been obviated by the growing belief that drainage is unnecessary and by the increasingly good results secured with chemotherapy and antibiotics.

#### IRRIGATION OF THE PERITONEAL CAVITY

Purulent exudates should be removed from the peritoneal cavity with the suction apparatus particular care being taken to introduce the tip into the pelvis and other dependent cavities. The maneuvers should be very gentle. Nonpurulent exudates have a high protective value and should not be disturbed. A plastic peritonitis also should not be disturbed. The experimental work of David and Sparks (cited by Orr) has shown that this type of peritonitis if well developed almost completely prevents the passage of colon bacilli from the peritoneal cavity into the lymphatic or blood streams.

Attempts to cleanse the peritoneal cavity by the introduction of saline solution and disinfectants are usually unsuccessful and may be extremely dangerous. The introduction of peptone broth as practiced by Brun and Feagles and of 70 per cent ethyl alcohol as practiced by Behan is of historic interest only since the development of modern chemotherapy and antibiotic therapy.

#### DRAINAGE OF THE ABDOMINAL WALL

In some cases in which drainage of the peritoneal cavity does not seem indicated drainage of the wound may be a wise precaution especially if the patient is stout or if the appendix is gangrenous or has been ruptured in the course of its removal. If a leaking appendix is smeared over an open wound which is then tightly closed infection is very

risk that nature will anticipate the surgeon and that rupture will occur internally (p 206)

When the patient is on the operating table and is fully anesthetized it is well to re examine the abscess to determine the point of maximum fluctuation so that the incision may be made over it When appendectomy would add little or nothing to the patient's load it should be performed both because there is no logic in leaving a leaking focus in the peritoneal cavity and because of the necessity of later secondary removal The appendix should not be removed however if one is dealing with a primary appendiceal abscess in which it forms part of the abscess wall or if its removal would involve the breaking down of adhesions

Drainage of a residual abscess that is readily accessible involves little more than incision over the fluctuant area removal of the purulent fluid by suction and insertion of a soft rubber tube or rubber dam drain about which the incision is loosely closed

A fully localized appendiceal abscess walled off by adhesions is almost or entirely extraperitoneal and there is never any excuse for invading the peritoneal cavity in draining it The cavity is entered with a blunt instrument usually Mayo scissors, at a point as distant as possible from any presenting loop of bowel and pus is removed by suction Flexible retractors of different widths are used to expose the cavity and a sterile light if necessary is introduced to be sure that all secondary loculations are opened up Drainage is carried out in the usual manner

An appendiceal abscess in the iliac fossa is evacuated through an incision in that region A retrocolic abscess in its early stages can be attacked through a vertical incision in the right lumbar region though in its later stages it must be approached along the posterior surface of the liver or treated as a subphrenic abscess

When a walled off abscess lies some distance from the parietal peritoneum the first difficulty is to recognize it the second is to drain it by a method that will not contaminate clean peritoneum Such abscesses must be thoroughly walled off by gauze collar dams before any attempt is made to open them Then a small opening is made at the point of greatest fluctuation and the suction tip is introduced by this method the greatest part of the pus can be removed with perfect safety since the tube blocks the opening All manipulations are conducted with the greatest gentleness for fear of rupturing the abscess After the cavity is emptied a drainage tube is introduced and brought out in the usual way Some surgeons instead of using this plan prefer to create a natural channel to the surface by placing a gauze pack about the abscess

## DRAINAGE OPERATIONS ON THE INTESTINE

Drainage operations on the intestine have lost their usefulness since the introduction of nonsurgical constant intestinal decompression which is much more efficient. Cecostomy still has a limited field of usefulness however in cases in which because of the condition of the cecum the appendiceal stump may be difficult to manage and ligation and inversion seem unsafe. A few surgeons occasionally advise it for other purposes. DeCourcy, and Dorrance and Neilson consider it an effective method of placing fluid in the intestine in the part in which it is best absorbed and the latter authors consider it a particularly valuable route for the administration of fluid in children.

The simplest method of performing cecostomy is to carry out appendectomy by the preferred technique except that two pursestring sutures (one within the other) are applied. Then instead of invagination of the stump a soft rubber tube (a de Pezzer catheter is satisfactory) is inserted in the opening before the pursestring sutures are tied. The cecal opening is sutured to the walls of the abdominal wound which is closed around the tube. The tube is sometimes brought out at the lower end of the incision but it is better to bring it out through a stab wound to lessen the risk of infection. The catheter is left in place until it separates of itself which is usually between the seventh and tenth days. Some surgeons prefer to place the enterostomy elsewhere in the bowel but this seems an unnecessary prolongation of the procedure in a patient in whom the mere performance of enterostomy is indicative of a serious condition.

The high jejunostomy formerly advocated by Wilkie and the anastomosis between the distended jejunum and transverse colon formerly advocated by Sampson Handley in addition to cecostomy are now merely of historic interest. The object of the latter procedure was to provide for a reflux flow of intestinal contents from the transverse colon along the ascending colon to the cecostomy.

## MANAGEMENT OF PRIMARY AND RESIDUAL ABSCESES

In opening a primary appendiceal abscess or a residual abscess following appendectomy the same general principles are followed as are applicable to similar infections anywhere else in the body. The evacuation of pus is always justified and should not be delayed too long even if expectant treatment is being deliberately employed because of the

which are opened within the cavity to enlarge the initial puncture. The finger is then inserted and worked around freely within the sac. If instruments are introduced they should be guided by the finger and the dissection should be kept as near the midline and the posterior uterine wall as possible. If necessary the abdominal hand can be used to push the abscess gently down into the vaginal canal while the pelvic hand makes the puncture but after the cavity is once opened no further pressure is applied and irrigation is not practiced. After the bulk of the pus has drained out a T shaped rubber drainage tube is carried into the depths of the cavity and is fastened to the posterior vaginal wall by one or two chromic catgut sutures.

Some authors have emphasized the wisdom of exploring the infected area from the abdominal side while it is opened from below so that clear visualization will prevent injury to the small intestines whose natural habitat is the pelvic cavity and which may encircle the abscess or form part of its wall. The bladder is not likely to be opened during colpotomy if catheterization is performed beforehand and the use of an aspirating needle as a guide in making the incision is an additional precaution.

#### MANAGEMENT OF SUBPHRENIC SPACE INFECTIONS

Although there has been considerable discussion over the route of approach to subphrenic space infections there is little need for it. The trans serous approach it is true is technically the easiest but it violates the principles of sound surgery in that it requires drainage of an infected area across an uncontaminated pleural or peritoneal cavity. Aside from their other advantages extraperitoneal operations are entirely free from this fundamental objection. The retroperitoneal operation was described by Nather and Ochsner and the preperitoneal method by Clairmont and Nather.

From the standpoint of mortality all the advantages are also on the side of extraperitoneal operation. In 1932 Flynn collected 313 surgical cases of subphrenic space infection handled by 61 surgeons; the mortality for the 275 cases operated on by the transpleural and transperitoneal routes was 41.0 per cent and for the 38 cases operated on by the extraperitoneal route 18.4 per cent. In 1942 surgical cases collected from the world literature by Ochsner and DeBakey in 1938 the mortality for transpleural drainage was 36.2 per cent for transperitoneal drainage 35.1 per cent and for retroperitoneal drainage 20.8 per cent. The authors' personal mortality for retroperitoneal drainage was 10.8 per cent (4 deaths in 37 cases). There would seem no doubt therefore that the

and conducting it to the external wound. The pick shortly becomes surrounded by adhesions and the pus finds the graze and thus makes its way to the surface. A better method is to remove the pick after a sufficient time has elapsed for the formation of a channel from the abscess to the exterior and incise and drain the abscess through this channel by the usual methods.

#### MANAGEMENT OF PELVIC ABSCESSSES

Residual pelvic abscesses are very satisfactorily handled by proctotomy or colpotomy and some authorities believe that these operations are also suited for the management of primary abscesses. The Charity Hospital figures do not support the latter point of view. When the operations were performed as primary procedures the mortality was prohibitive there being 11 deaths in 26 cases in contrast to no deaths in the 46 (of 122) residual cul de sac abscesses drained by the same methods.

Both operations are carried out in an exaggerated lithotomy position after the bladder has been emptied by catheter. For proctotomy a retractor or small vaginal speculum is introduced into the anus. Following identification of the abscess by cautious aspiration of pus the point of fluctuation on the rectal wall is penetrated by steady gentle pressure with the closed tips of a pair of scissors. As soon as the puncture is made the blades are widely opened. A drainage tube is stitched into the abscess cavity and fastened at the anal margin by a single stitch. Incision through the rectum carries little risk to loops of bowel since their contents are gaseous and they float above the fluid in the cul de sac. Reinfection from the rectal contents is a theoretic possibility but practically closure of the rectal opening is likely to occur too rapidly rather than to present a later problem of closure in an infected field.

When colpotomy is performed a weighted speculum is inserted into the vagina and the cervix is elevated by a vulsellum inserted in the posterior lip so that the posterior vaginal wall is fully exposed. Following aspiration a small crescentic incision is made across the posterior cervicovaginal junction as the posterior vaginal wall is pulled downward and held on tension by forceps. The incision runs to the lateral angles of the junction but should not be extended upward along the sides of the cervix. Bleeding is seldom of consequence since no large vessels lie in the cervicovaginal space and the highly vascular para-cervical area is not entered at all.

The abscess exposed by the incision is punctured with closed scissors

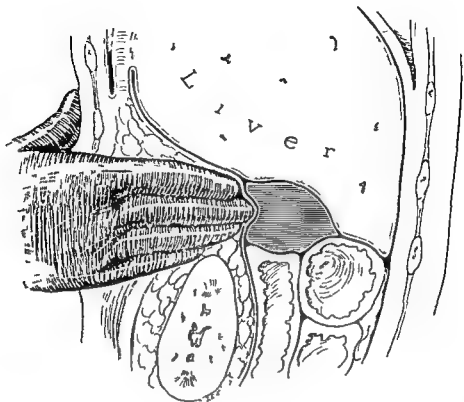


Fig 65 Technique of Nather Ochsner operation for subphrenic space infection  
Palpation of abscess in subhepatic space prior to incision

1 Incision over and down to the twelfth rib to within 4 cm of the midline posteriorly Subperiosteal resection of the entire twelfth rib

2 Transverse incision of the musculature below the bed of the resected twelfth rib at the level of the first lumbar spinous process followed by dissection down to the renal fascia The spinous process should be marked before operation so that the pleura will not be opened The renal fascia is recognized as a smooth shining fibrous layer through which the renal fat can be seen

3 Blunt separation of the musculature from the renal fascia for a short distance above and below the wound Elevation of the diaphragm and pleura by broad dull retractors Blunt separation (by the index finger) of the peritoneum continuous with the renal fascia from the under surface of the diaphragm The separation can be carried as far as the dome of the liver if it is necessary to reach an abscess high on the



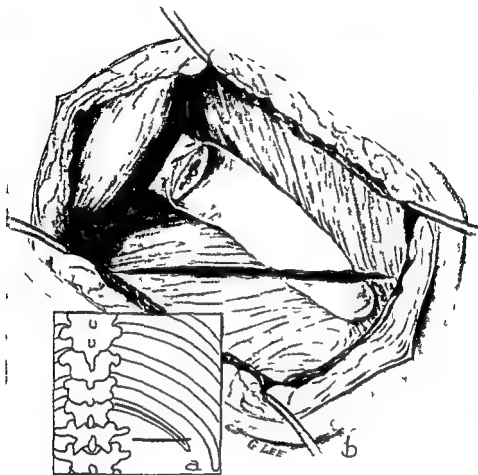


Fig 64 Technique of Nather Ochsner operation for subphrenic space infection a skin incision over twelfth rib and incision at level of spinous process of first lumbar vertebra after resection of twelfth rib Note that the deeper incision is not in a line with the skin incision As frequently pointed out by Ochsner and his associates the relationship of the costophrenic reflection of the pleura to the twelfth rib and to the spinous process of the first lumbar vertebra varies considerably but the pleural reflection is always above a transverse line at the level of the spinous process of the first lumbar vertebra Attention to the level of the incision prevents entrance into and contamination of the pleural cavity b transverse incision through twelfth rib at level of spinous process of first lumbar vertebra Thus incision divides the serratus posticus inferior and latissimus dorsi muscles and the attachment of the diaphragm (After Ochsner)

results as well as the rationale of the extraperitoneal route should advise its general use

The operation advocated by Nather and Ochsner is carried out as follows (figs 64-6)

upper posterior space. If pus is obtained the abscess is opened by breaking through the peritoneum over it with the finger. Abscesses in this location and in the subhepatic space are drained by the introduction of large rubber drainage tubes and iodoform gauze drains into the cavities. Irrigation is not practiced.

7 If empyema exists the pleural cavity may be drained by large rubber tubes after simple incision of the pleura above the diaphragm. Further rib resection is not necessary.

If abscesses in the retroperitoneal and retrocecal areas are suspected exploration can be carried out by extension of the skin incision downward and forward in the direction of the anterior superior iliac spine and separation of the muscles in their respective directions.

The advantages of this technique according to Nather and Ochsner are

1 Both the suprahepatic and the infrahepatic space can be explored and drained by a single procedure.

2 The unaffected pleura and peritoneum are not contaminated.

3 Only a short functionless structure the twelfth rib is resected and the wound is surrounded by soft parts which fall together as soon as the drainage tubes are removed with prompt obliteration of the cavity and material shortening of convalescence.

4 The abscess is drained in its most dependent portion.

■ The retroperitoneal retrocecal and perinephritic spaces can be explored if necessary by extension of the incision downward and forward. If necessary the entire subphrenic region can be drained by combining this operation with the preperitoneal operation devised by Clairmont and Nather.

6 Shock is reduced to a minimum since only one body cavity is entered.

In the preperitoneal operation (fig 67) the anterior suprahepatic space infection is approached through a subcostal incision. The dissection is carried down through the posterior rectus sheath. The peritoneum is then separated by blunt dissection from the diaphragm until the abscess is encountered. Thereafter the operation is conducted as in the retroperitoneal method.

Clute in a discussion of Faxon's paper on subphrenic abscess stated that he occasionally used a two stage procedure in the first stage of which 11 cm. or more of the tenth rib are removed in the midaxillary or postaxillary line the diaphragm is sutured to the parietal pleura and the wound is packed. The pleural cavity becomes walled off within 24 to 48 hours and the large exposure simplifies exploration of the subphrenic

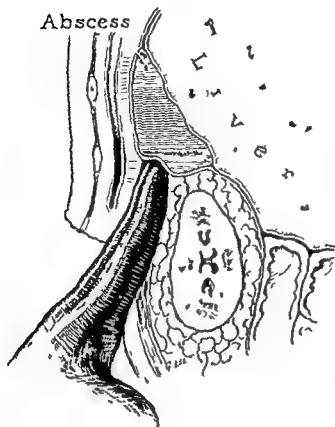


Fig 86 Technique of Nether Ochsner operation for subphrenic space infection. Palpation of abscess in right posterior superior space prior to incision

hepatic surface. If an abscess is present the peritoneum is edematous and is more readily separated than normal peritoneum. If the finger follows the direction of the diaphragmatic fibers which run parallel to the ribs dissection is also simplified.

4 Aspiration of the pleural cavity above the diaphragm if empyema is believed to be present. The finger is kept in the subphrenic space between the dissected peritoneum and diaphragm so that the aspirating needle can be guided and prevented from puncturing the diaphragm.

5 Exploration of the subhepatic space by the aspirating needle. If pus is obtained the needle is withdrawn and the area is tamponaded with gauze until after exploration of the suprahepatic space. The point of election for abscess formation in this location is between the visible right lower border of the liver and the upper pole of the right kidney.

6 Exploration of the suprahepatic space by means of the curved needle advocated by Clairmont for exploration of the upper surface of the liver. The needle is directed well posteriorly since the site of election for postappendiceal abscess in the subphrenic space is the right

## (VII)

# *The Therapy of Acute Appendicitis and Its Complications*

## II ADJUVANT MEASURES

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### UNCOMPLICATED ACUTE APPENDICITIS

#### PRE OPERATIVE CARE

In the uncomplicated case of acute appendicitis only routine pre operative preparation is required. It is assumed that examination of the chest, determination of the blood pressure, urinalysis, and blood count will have been carried out in the course of diagnosis. If the patient has a chronic disease such as cardiac or renal disease or diabetes, it is well that he be examined by an internist (preferably his own physician—if he has one—who is familiar with his condition) and the consultant's recommendations in regard to the administration of digitalis or insulin and similar matters should be followed. Undue time, however, should not be taken for elaborate preparation. In the absence of indications for delay, which do not usually exist in the uncomplicated case of acute appendicitis, the patient's best interests are served by prompt appendectomy.

Immediate pre operative preparation consists of the hypodermic administration of morphine sulfate and atropine, the dosage being calculated on the basis of the patient's size and age. If there has been no vomiting, phenobarbital or some similar drug can be used by mouth; otherwise, scopolamine or some similar drug can be administered by hypodermic. These are chiefly matters for the anesthetist to decide, but pre operative sedation, which will lessen or eliminate the discomforts of the first few hours after operation, is always desirable in the absence of contra indications.

#### POSTOPERATIVE CARE

Postoperative care in uncomplicated acute appendicitis is as simple as pre operative care and should not be made difficult. By the third day after operation, and frequently much earlier, the usual patient is in good

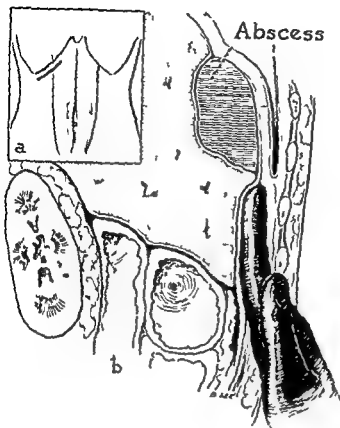


Fig 67 Technique of Clairmont Nather operation for anterior subphrenic space infection a location of subcostal incision b extraperitoneal dissection of peritoneum from under surface of diaphragm with finger to reach abscess

cavity at the second operation. This method obviates the possibility present in the Nather Ochsner operation of injuring the pleura which sometimes runs to the level of the twelfth rib and a little beyond it.

McWhorter described another method of extrapleural drainage for cases in which the pleural reflection is so low that it cannot be pushed up without resection of additional ribs and there is risk of accidentally opening the pleural cavity.

needed longer than 24 or 36 hours and may not be needed at all. Codeine sulfate after the first 12 hours is often as effective as morphine and should be used in preference to it if sedation is necessary. When peristalsis begins to return discomfort is due to other causes and should be handled by other measures.

*Fluid and Food Intake* No fluid should be permitted to a patient who is nauseated and vomiting until both nausea and vomiting have ceased and no fluid should be swallowed by a patient who has had an inhalation anesthetic even if he is not vomiting for a minimum of 4 to 8 hours after operation. There is no objection however to rinsing the mouth with water at intervals if the fluid is not swallowed. After nausea ceases tap water is given at first in sips later in gradually increasing quantities if it causes no disturbance. Small pieces of ice are also permitted but cold fluids are undesirable at this time. Hot sweetened tea is excellent. There is no indication for the administration of fluid by vein or other parenteral routes to a patient in good condition who is not vomiting and who is likely to be returned to full fluids within a few hours.

Until normal peristalsis returns usually within 36 to 72 hours after operation the intake is limited to sweetened drinks, thin broths and soups and other nonresidue fluids. Milk which leaves a residue should not be taken at this time. After peristalsis is evident the patient is put on soft diet and is returned to full diet as rapidly thereafter as possible. No special dietary regimen is necessary but it should be remembered that an inactive person confined to bed who is in good condition does not need unduly large amounts of food though it is important that the fluid intake be adequate.

*Intestinal Function* When peristalsis begins to return the patient may complain of gas pains. They are seldom severe and seldom last long in a patient who is progressing smoothly. They are best relieved by the use of the rectal tube and the application of heat preferably by a heat tent the temperature of which can be controlled. Complications are unlikely to develop in a patient in whom peristalsis returns within 48 hours after operation. If careful observation is supplemented by auscultation it will be found that returning peristalsis is usually apparent first in the left lower abdomen then in the right upper abdomen and finally in the mid right abdomen.

If the bowels do not move voluntarily when the patient resumes his usual diet it is better to permit another day or two of constipation than to use enemas or laxatives which sometimes excite a dangerous amount of intestinal activity and may even blow out the suture line and cause an internal fecal fistula (p. 236). Orr's experimental evidence indicates

condition and needs only routine nursing care if that. Throughout the postoperative course however he should be carefully observed for unexpected complications are possible in any case. With this proviso only simple therapeutic measures are required and no others should be instituted.

*Immediate Postoperative Measures* Wet clothing is changed in the operating room and the patient is returned to the so-called ether bed the preparation of which is routine in most hospitals. The heat regulating mechanism of the body is disturbed by anesthesia and operative manipulations and the temperature frequently falls to subnormal levels so that external heat may be necessary even in summer. It is unsafe to leave hot water bags in the bed with an unconscious patient unless the water is only moderately hot serious burns may be sustained otherwise for which the hospital may be held legally liable. Electric pads should never be used. It is unwise as well as unnecessary to keep the patient in the ether bed too long for excessive perspiration will cause an undesirable loss of body fluids.

The patient who has had an inhalation anesthetic should be kept under constant observation until consciousness has completely returned to see that he does not fall out of bed or injure himself by sudden movements. Vomiting is not usually troublesome and all that is necessary is to keep the head turned to the side if it should occur so that vomitus is not aspirated into the respiratory passages to guard against obstruction of the air passages by other accidents such as swallowing the tongue and to provide other simple nursing care.

*Posture* In the uncomplicated case of acute appendicitis there is no indication for any special posture. The supine or lateral supine position should be used until consciousness has returned. Then the patient may have one or two pillows under his head or may be propped up if he wishes to be. It is an essential part of the nursing care to see that the patient does not remain for long periods in the same position particularly if he is advanced in years for even in uncomplicated cases atelectasis hypostatic pneumonia and other respiratory complications may develop (p. 242). The position of an unconscious patient is changed by the nurse. A conscious patient is instructed to change his position at regular intervals and is observed to be sure that the instructions are carried out. The taking of several deep breaths and coughing at hourly intervals are also insisted on.

*Sedation* After the effect of the preoperative sedation has begun to wear off morphine sulfate is used in sufficient quantities to keep the patient comfortable but is withdrawn as rapidly as possible. It is seldom

it is not disturbed until the sutures are removed which is usually on the seventh postoperative day. A small dressing secured with fresh adhesive tape is then applied to the incision and is changed as necessary until complete healing has occurred.

**Resumption of Activity** A patient who has been operated on through a McBurney incision is permitted to sit up in a chair on the third day. If another type of incision has been used a few more days of complete bed rest are advisable. Ambulation is then resumed gradually and the patient is permitted to go home as soon thereafter as is convenient. If convalescence has been smooth the normal routine of life with restriction of excessive physical activity is possible within 2 to 3 weeks of operation.

**Early Postoperative Ambulation** The conservative postoperative routine represents a considerable shortening of the former postoperative course but within the last few years numerous surgeons have abbreviated it even more by not only permitting but urging patients to get out of bed shortly after operation. Ries in 1899 seems to have been the first to advocate this plan and it was occasionally practiced by European and American surgeons for the next decade. Boldt in 1907 stated that he had personal knowledge of more than a thousand patients including his own and Ries patients who had been thus treated without serious consequences and with generally good results.

Articles on the subject continued to appear in the European literature but in the American literature almost nothing else was written on it until 1941 when Leithauser and Bergo published a report of 436 surgical cases treated by this method. Since that date numerous other reports have appeared almost uniformly favorable and the practice has become increasingly popular. Nelson's study from the Charity Hospital of Louisiana at New Orleans in 1944 covers 429 operations involving incision of the abdominal wall; in all instances the patients walked within 72 hours and in the majority of cases they walked on the day of operation or on the following day. The single fatality in the series was caused by cerebral thrombosis. Two of the 3 partial wound disruptions occurred in patients in whom absorbable suture material had been used and in whom early ambulation was permitted in error. Two incisional hernias, both small, were observed within a 2 year follow up period and there was 1 instance of recurrent incisional hernia in a patient in whom the original repair had not been adequate. There was a striking absence in the series of such complications as phlebitis, embolism, peritonitis and urinary tract infection. Three hundred seventeen of the 429 cases were appendectomies.



that solutions usually employed in enemata do not stimulate peristalsis in the small intestine while liquids given in quantity by this route are difficult to expel and may cause considerable discomfort. Mineral oil can safely be used after the third or fourth postoperative day and a low enema given with very little force may be used by the sixth or seventh day in the occasional case in which it is necessary.

*Urinary Function* Urinary retention even in the patient who is otherwise entirely normal may be an annoying feature of convalescence. The bladder is emptied before the patient goes to the operating room by catheterization if preoperative sedation has already been effective and voiding is seldom a problem until 8 or 10 hours after operation because the intake of fluids is restricted in the interval. A patient who does not express a desire to void after this time should be examined to determine whether the bladder is full. If the outline of the bladder is not visible on the abdomen as it usually is not and if it cannot be made out by palpation percussion which produces a flat note may be useful. The diagnosis of urinary retention can be confirmed if necessary by rectal examination.

Every endeavor should be made to see that the bladder does not become too full for the effects of anesthesia particularly of spinal analgesia and the use of sedatives may blunt the patient's sensibilities so that urinary retention may be present without his realization. Crude measurement of the amount of urine voided for the first few days after operation is a wise plan even in the normal case to be sure that the bladder does not contain residual urine as well as to check the fluid intake against the output.

A patient who has difficulty in voiding after operation should be treated by simple measures such as warm applications and irrigations before catheterization is resorted to. Often the temporary assumption of the sitting position produces good results. If however these measures are not successful catheterization though undesirable should be resorted to without too much delay for prolonged overdistention of the bladder is even more undesirable. It should be carried out with strict attention to asepsis though there is no reason for the instillation of antiseptics into the bladder unless the procedure must be repeated over a period of days. The patient should be encouraged to attempt to void at regular intervals after the first catheterization and the procedure should not be carried out a second time until the simple measures already outlined have again been resorted to.

*Care of the Wound* The wound is dressed in the operating room and in the absence of local pain fever or other evidences of infection

who tolerate dehydration and toxemia very poorly As Murphy expressed it in 1904 80 per cent of the patients submitted to appendectomy for acute appendicitis need little if any special attention after operation 10 per cent need very close watching and 10 per cent need the exercise of the greatest skill if they are to recover That observation is still generally true

The measures to be described in this chapter are applicable to patients treated conservatively without immediate operation as well as to those treated surgically and no special distinction will be made between their use in surgical and in nonsurgical cases Many of the same measures furthermore are applicable in both pre operative preparation and postoperative care and they will be described under single headings to avoid unnecessary repetition

There should be no undue delay in the preparation of a patient with rupture of the appendix and general peritonitis for surgery On the other hand a dehydrated toxic patient should not be operated on until gross dehydration has been corrected by the use of one or more infusions and perhaps a transfusion Thereafter these measures should be used according to special indications supplemented by blood blood chemical and other studies It is peculiarly unsafe to operate on patients with very low blood pressure especially if cardiac disease is present also and on patients in toxic delirium until some degree of rehabilitation has been achieved

#### GENERAL MEASURES

*Posture* The patient after operation lies in whatever position he finds most comfortable provided that he changes it frequently or that it is changed for him Fowler's position is no longer believed to possess the special advantages which it was once supposed to have and is thought to be actually undesirable because it impedes circulation and therefore invites thrombosis in the veins of the legs If for any reason it should be used in a special case this possibility should be borne in mind

*Application of Heat* External heat is applied over the abdomen preferably by a heat tent the temperature of which can be controlled so that there is no danger of burning the skin Compresses and similar methods are disturbing to the patient require continuous nursing service which is not always available and possess no special advantages

The obvious reason for using heat in the treatment of peritonitis is that it adds to the patient's comfort It has also been suggested probably with reason that it influences peristalsis and improves the blood supply to the peritoneum Because it reduces the amount of blood in the

Nelson who emphasized that early ambulation must not be employed unless nonabsorbable (cotton or wire) sutures are used outlined his own procedure as follows

As soon as the patient has fully recovered from the effects of whatever anesthetic has been used the bed is sharply tilted, so that the head is elevated. After this position has been maintained for a time the bed is leveled and the patient assumes a sitting position on the side of the bed with the feet resting on a chair while he breathes deeply and coughs frequently. After a second period of recumbency with the head of the bed again sharply elevated he is assisted to stand and is conducted to the bathroom where the bladder can practically always be emptied without difficulty. If his condition is good he is permitted to sit up in a chair for a time before returning to bed. Thereafter he rises at will goes to the bathroom fetches his own drinks sits on the sun porch and is active within reason in proportion to his desires.

The rapidity with which complete ambulation is achieved varies with the individual case but ordinarily there is no particular delay between the various steps described though greatly debilitated patients may find it necessary to proceed slowly. It is important that all initial activities be supervised by the nursing staff and that excessive fatigue be prevented. No patient is made to walk or to get out of bed against his will. In open wards however the majority stimulated by the example of other patients are eager to be active themselves.

The advantages of early ambulation listed by Nelson are much the same as those listed by other proponents of the practice. They include a lowered incidence of postoperative complications particularly pulmonary and vascular complications a lowered incidence of nausea vomiting and abdominal distention early return of normal function of the bladder and bowel maintenance of normal muscle tone psychologic improvement in morale and mental status acceleration of convalescence and earlier return of working ability and economic savings to both the patient and the hospital. In spite of these advantages it is well to bear in mind that early ambulation is not a panacea and that its imprudent use in cases in which it is not indicated will certainly lead to trouble.

#### COMPLICATED ACUTE APPENDICITIS

The patient with complicated acute appendicitis is often a problem both before and after operation and whether or not he recovers depends almost as much upon the quality of preoperative and postoperative care he has received as upon the removal of his appendix. This is particularly true of very young children and of persons advanced in years.

that its use was chiefly confined to hopeless cases in which there was literally nothing else to do (p 362)

McBurney long ago pointed out the risk of operating on patients with distended intestines. He wrote

Are there any contra indications to this operation in a clear case of appendicitis? I think there are. Very great abdominal distention which in a given case might probably be relieved by a few hours treatment would lead me to delay the operation for expulsion of intestine is a very serious obstacle to the proper completion of the operation without risk.

The introduction of suction drainage or constant decompression of the intestinal tract by Robertson Ward and its popularization by Wangenstein has completely changed this picture. Ward states that by the use of this method the mortality of acute appendicitis at the University of California Hospital has been reduced from 58 per cent to 3 per cent and others have shown equally good results. The recent reduction apparent in the mortality of complicated acute appendicitis at the New Orleans Charity Hospital (p 422) can be largely attributed to the use of constant suction plus the more consistent and more rational use of replacement therapy.

Constant suction through an indwelling nasal tube removes the liquid content of the intestine and quite as important also removes the gas and swallowed air which are partially responsible for intestinal distention. The method is of the greatest value when it is begun promptly before distention has assumed massive proportions. Distention is the indication for its use; not nausea and vomiting; they do not necessarily accompany distention for the response to pathologic processes differs in the individual patient according to the capacity of his stomach, the sensitivity of his nervous system and other factors. Another reason for the prompt introduction of the tube is that its onward passage is dependent upon gastric and intestinal peristalsis; if peristalsis has completely ceased it is difficult to pass the tube into the small intestine or very far into it and maximum benefits cannot be expected.

Excellent as are the results of intestinal decompression it should be remembered that the method has definite disadvantages when it is used over long periods of time. The continuous removal of gastric and intestinal secretions, especially chlorides, disturbs the fluid and electrolyte balance of the body as well as the nutritional balance and the curious situation develops that replacement therapy is required because of a therapeutic method as well as because of a primary disease.

Other complications are also possible as Berger and Achs pointed out

splanchnic area it has a favorable effect on intestinal distention. Finally its vasodilating effect perhaps lessens the danger of thrombophlebitis.

The ice bag is no longer used in the treatment of peritonitis. Aside from the fact that it does no good its anesthetic effect may mask symptoms and the ischemia which it causes may be responsible for wound infection.

**Sedation.** The use of morphine to make the patient comfortable after operation is a practice of long standing but only within recent years has it been realized that in addition it has a beneficial effect on the intestinal tract. Ileus is a natural protective state in peritonitis but it is potentially a dangerous state and morphine which improves intestinal tone without at the same time causing violent intestinal contractions adds to the patient's mental and physical comfort.

Morphine should be used in dosages appropriate to the patient's size and age at regular intervals. Its use in older persons should be strictly limited. Orr's experimental studies have shown that the maximum clinical benefits including stimulation of the tone rhythmic contraction and to some degree the peristaltic waves of the small intestine can be secured only when the drug is given at regular intervals and in sufficient dosage to produce continuous narcosis. A single dose is effective only for a maximum of 6 hours. The administration in full dosages should be continued until there are signs of returning peristalsis or until decreased respirations (14 to 16 per minute) or cyanosis indicate that too much is being given. Then the dosage is decreased or the intervals between the injections are lengthened and the drug is discontinued entirely when it is evident that peristalsis has been fully restored. Gas pains (p. 295) should not be treated by sedation.

#### CONTROL OF INTESTINAL DISTENTION

Ileus as has just been pointed out is part of nature's protective mechanism but an extreme degree of intestinal distention is always evidence of an advanced disease process. It seriously complicates any operative procedure and for mechanical reasons if no other should be reduced before surgery is attempted.

The old methods of treating ileus were by gastric lavage and enterostomy. The former is of only temporary benefit and such effects as it achieves are limited to the stomach which is seldom involved in the process until the disease is hopelessly advanced. Enterostomy was never a particularly rational method of treatment of ileus since it decompresses only the immediately adjacent segment of bowel and it is small wonder



Fig. 69A Insertion of Miller Abbott tube  
A Tube in stomach



Fig. 69B Insertion of Miller Abbott tube  
B Tube in duodenum



Fig. 69C Insertion of Miller Abbott tube  
C Tube in small bowel

in a report of what is apparently the only recorded instance of perforation of the small intestine by the Miller Abbott tube. In this case the tube which was *in situ* for 12 days was reportedly halted at the same point in the intestine. At operation perforation of the wall of the ileum was found to have occurred immediately proximal to the point of obstruction which was also the point at which the tube had halted.

These authors in a review of the literature in connection with their report collected numerous instances of accidents following intubation including esophageal strictures reported by Vinson, injuries to the larynx reported by Mahon, by Morrison, by Ighuer and Molt, and by Kaufman, Serpico and Mersheimer, injury of the arytenoid cartilages reported by Wangenstein, and rupture of the stomach reported by Glissman, by Wolf, and by Lemmon and Paschal. Obviously when intubation is employed frequent roentgenologic observations must be carried out and when it is prolonged careful daily inspection of the larynx is necessary.

**Mechanism of Intestinal Decompression.** The principle of gastrointestinal decompression is a much more important consideration than the exact apparatus used. The latter may be as simple or as complicated as the surgeon wishes. By the Wangenstein method a Levin tube, the distal end of which is perforated for 10 or 11 inches, is passed through the nose into the stomach and duodenum and is then connected with a siphonage apparatus which exerts a constant suction of about 75 cm. of water. The contents of both the stomach and the duodenum can be drained off by this method.

The Miller Abbott double lumen tube permits drainage down to the ileocecal valve but decompression by this method does not differ in principle from the method introduced by Ward and by Wangenstein. One lumen of the tube controls a balloon by the inflation and deflation of which the tube advances along the intestinal tract; the other lumen is used for the aspiration of fluids and gases.

Considerable practice is necessary before the surgeon becomes adept in the introduction of this type of tube. Although variations have been introduced and may be necessary in individual cases, the method described by Abbott and Johnston, which is based on the original description by Miller and Abbott, is satisfactory in most cases.

The type of tube now commercially available consists of two lumina: a larger lumen for the aspiration of gastrointestinal contents and a smaller lumen for the introduction of air into the balloon. This tube can be introduced into the stomach through the nose without being brought out through the mouth, as was necessary with the first type of Miller Abbott tube. Introduction is preferably accomplished under the fluoro-

grams or fluoroscopic examinations now required to localize the Miller Abbott tube accurately

Harris suggested the use of liquid metallic mercury in the rubber balloon of the Miller Abbott tube to facilitate its passage. When he reported the method in 1943 he had used it in 19 cases over a 2 year period with no complications or failures and with considerable shortening of the time of insertion. In the last cases of this series the balloon had not been inflated with air the weight of the mercury in itself carrying the tube down the gastrointestinal tract. In 1945 Harris reported the use of a mercury weighted calibrated single lumen tube in 18 obstructions of the small bowel. In this tube the entire lumen is devoted to purposes of drainage the diameter of the lumen is larger than the diameter of the lumen of the Miller Abbott tube which is used for drainage though the tube itself is smaller than the Miller Abbott tube.

Since Harris original report Herrera Millet and Lawrence have reported the use of a tube similar to Harris in 30 cases with success in 26 and Cantor Kennedy and Reynolds have reported the treatment of 200 patients with successful passage of the tube in 192. In 2 of Harris cases and in 2 reported by Herrera and his associates (who now use a double thickness bag) rupture of the bag occurred and mercury spilled into the gastrointestinal tract. Liquid metallic mercury however is fluid and flexible and in each instance it passed rapidly through the intestinal tract and was excreted without harmful effects.

The most recent report on the Harris modification of the Miller Abbott tube was made by Harris and Gordon who reported its use in 100 instances (86 patients). The complications in these cases included coiling of the tube in the stomach failure to pass the pylorus inability to withdraw the tube rupture of the bag and osmotic swelling of the rubber bag containing the mercury. As a result of this experience certain changes in technique were recommended. The tube has been reduced in length from 9 to 6 feet. The former advice that it be introduced at the rate of 1 inch every 10 minutes is rescinded and a strict hands off policy is followed. When it has entered the nares for a distance of 3 feet a roentgenogram is taken. If the patient is able to stand up or to walk around the force of gravity assists the introduction of the tube. If he is confined to bed he lies on the right side in a high semi Fowler position. When this procedure is followed the tube never coils in the stomach. The tube is passed beyond the pylorus for a distance of not more than 2 or 3 feet. This is a sufficient length for the intestine to be plicated on the tube accordion fashion and decompression of the entire small bowel is thus accomplished.



scope. The tube is passed through the nose and is swallowed just as is the usual Levin tube. When the tip is seen to have reached the mid portion of the duodenum a volume of air (about 30 cc.) is injected into the balloon while constant suction is maintained on the larger tube. As gas and fluid are sucked out of the intestine the intestinal walls contract and having regained their normal propulsive movement force the balloon ahead. The suction collapses the intestine in successive loops as the tip of the tube is advanced (figs 68A-C).

In some instances the tube can be introduced through the full length of the small intestine to the ileocecal valve within a few hours. In others introduction is more difficult and the procedure may occupy as long as 24 to 72 hours. When the tip of the tube has reached the third portion of the duodenum its progress is usually automatic thereafter. The tube is marked at 6-inch intervals so that the speed of its descent can be gauged. If it is swallowed too rapidly a loose coil may form in the stomach and further passage will be prevented. It may then be necessary to remove the tube and reintroduce it. Insertion of the tube under the fluoroscope and checking of its rate of advance and position by X-ray or fluoroscopic observations simplify the use of this method though moving a seriously ill patient has its obvious disadvantages.

McLennan recommends abdominal auscultation carried out simultaneously with the production of noise within the balloon as a simple and effective method of locating the position of the tip of the Miller Abbott tube. The observer using a stethoscope determines the point of maximum intensity of the sound while an assistant alternately inflates and deflates the balloon with air. If the tip is within the intestine a definite point will be identified beneath which the sound of air rushing in and out of the balloon seems almost to be in the observer's ears. If the balloon is still in the stomach the sound which will be audible across the epigastrium especially to the left is not so sharply localized. Moreover because of its presence in a larger viscus the sound takes on a resonant quality in the stomach which it does not have in the intestine.

The test is also satisfactory though localization is less accurate because of multiple openings in the tube when single lumen tubes with mercury bulbs are employed the suction tube instead of the balloon is then used for the production of the sound.

McLennan reports that he has used this test for more than 2 years and has found it satisfactory when checked by roentgenologic and other methods. Speed and accuracy naturally improve with practice. If the method proves generally applicable it will greatly reduce the time effort, and expense involved in the taking of the numerous roentgeno-

sary and if a serious need for them arises constant intestinal decompression is the safer plan

#### REPLACEMENT THERAPY

**Fluid Balance** In the healthy person living in normal environmental conditions the daily intake and output of fluids results in the maintenance of the water content of the body at an essentially constant level. The intake of fluid as such usually varies from 800 to 2 000 cc daily the higher intakes occurring in hot weather when loss by vaporization is great. Each gram of solid food ingested furnishes approximately 0.9 gm of water and from this source a person on a routine maintenance diet receives from 1 000 to 1 500 gm (900 to 1 350 cc) daily.

The intake of fluids in these amounts compensates for the following daily normal losses:

1. Loss by insensible perspiration from the body surface which amounts to approximately 1 000 cc.

2. Loss by sensible perspiration which varies with the environmental temperature and the total caloric expenditure. Generally speaking loss by this route is much greater in warm than in cold climates and during activity than during inactivity.

3. Loss by water of evaporation in exhaled air which amounts to approximately 300 cc daily.

4. Loss of water in feces which amounts to 150 to 250 cc daily.

5. Loss of water by urine the optimum daily output of which is at least 1 500 cc.

The loss of water of vaporization by way of the skin and lungs Maddock and Collier have pointed out can be regarded as having preferential rights on loss of water by the kidneys. In other words the amount of urine voided represents the excess of fluid in the body after other channels of fluid excretion have been satisfied. A person with normal kidneys the powers of concentration of which are exerted at their maximum must void at least 500 cc daily to rid the body of the average amount of waste material. The concentration of urine should not exceed 1 020 to 1 030 to provide for the approximately 50 gm of urinary solids which must be excreted daily. If the available fluid is reduced below 500 600 cc by reduction in the fluid or food intake or for other reasons the function of the kidneys is disturbed urinary constituents accumulate in the blood and uremia ultimately sets in if the fluid balance is not restored. Clinically the lack of fluid becomes apparent in the patients

In one case in Harris and Gordon's series persistent attempts to pull the head of the tube back through the ileocecal valve resulted in a tear of the bowel and the production of a localized abscess. It is now their policy when difficulty is encountered to cut the tube at the nares and permit it to be eliminated via the anus or, if the opening exists via colostomy. In the 7 cases in which this was done the tube was passed in from 24 hours to 10 days and the patients suffered no ill effects. The authors concluded that difficulties in removal of the tube occurred because of the increase in the amount of air or gas in the bag, the permeability of the rubber condom permitting the interchange of gases. They believe that if the bag is flushed with carbon dioxide before it is fastened secondary swelling will not occur.

Whatever method of decompression is used the drained fluid should be measured and a description of its appearance should be entered on the chart. The patient regardless of his condition is permitted to drink water and other fluids to satisfy thirst and keep the mouth moist; they do no harm since they are immediately aspirated. Intake should be measured and compared with the amount of fluid drained through the tube.

Intestinal decompression if the proper replacement therapy is employed may be continued over a period of days. As the patient begins to improve the tube is clamped for periods of 2 to 3 hours and functional improvement is estimated by comparing the intake of fluid during this period with the residual fluid removed by suction at the end of the time. If the amount of fluid aspirated is less than the intake the return of peristalsis may be assumed though the tube should not be removed until it is certain that improvement has occurred and is likely to be permanent for a second intubation particularly with the Miller Abbott tube is often more difficult than the first.

*Other Methods of Intestinal Decompression* Other methods of decompressing the intestine and restoring its tone need no extended consideration. Concentrated solutions of sodium chloride have occasionally been used on the basis of experimental evidence (Orr) but the general opinion is that it is more desirable to maintain the blood chlorides at a normal level.

Most authorities do not consider it wise to use drugs to stimulate active peristalsis during the acute stage of peritonitis since such measures disturb the protective mechanism which is at the basis of both natural and therapeutic methods of control. A few observers favor and have reported good results from the use of pitressin or prostigmine both prophylactically and therapeutically but such drugs are usually unneces-

only in clinical malnutrition but in a fall in the protein concentration of the blood which normally is 6-8 gm per cent children above 2 years have the same values as adults

Disturbance of the mechanism of exchange of water between the blood plasma in the capillaries and the fluid in the tissue spaces surrounding them results in damage to the capillary walls which become permeable to protein and permit its escape in the form of plasma. This condition though often associated with dehydration is not dehydration *per se*. The patient has adequate fluid in his body but it is in the wrong place. The resulting protein disturbance is aggravated by the use of water or salt solution which serves merely to wash more protein out of the injured capillaries. Incidentally it is not until protein deficiency has reached an advanced stage that it begins to become evident as edema.

*Disturbances of the Fluid and Chemical Balance in Complicated Acute Appendicitis* The patient with complicated acute appendicitis is dehydrated and his chemical and nutritional balance is disturbed for several obvious reasons. He has taken only minimal amounts of food and fluid since the beginning of his illness and has probably derived little if any benefit from what he has ingested because most of it has probably not been retained. He has probably lost additional body fluid because of vomiting or perhaps diarrhea. If he has been operated on he has lost blood at operation the amount in the absence of true hemorrhage often being more than the surgeon realizes. Baronofsky and his associates found that in 5 (presumably average) appendectomies the blood loss estimated by the gravimetric method ranged from 50 to 350 cc and averaged 258 cc.

The patient's loss of water by vaporization has probably been greater than normal because of the rather high temperature at which operating rooms are kept and the warmth of the linens used in the course of the operation. More fluid will be lost by this route if he is put into an ether bed and kept there an unduly long time. If he has been submitted to a drainage operation or to a cecostomy or if a fecal fistula or a wound infection develops fluid is also lost by these routes. Finally as has been pointed out constant intestinal decompression along with its benefits produces a constant loss of fluids and chlorides.

The loss of protein is likely to begin almost as soon as the patient becomes ill either because he is anorectic or because the normal intake is prevented by nausea or vomiting. There is a further loss at operation as the result of two mechanisms. (1) Blood is actually lost or blood plasma or exudate is spilled into the traumatized area. (2) Protein tissue breaks down after operation particularly if an infectious process is

state of dehydration in addition to being oliguric or anuric he presents a dry loose skin sunken eyes and a parched and furred tongue

The water which is absorbed in the body chiefly from the lower portion of the gastro intestinal tract enters the blood the interstitial spaces between the cells and the cells themselves When for any reason fluid intake is reduced and the body must begin to utilize its own water content the water from the blood and interstitial spaces is mobilized first and the water content of the cells last of all Losses of 2 and 3 per cent of body fluid are readily replaced A loss of 5 or 6 per cent is serious Losses of 15 to 20 per cent are likely to be fatal

*Chemical (Acid Base) Balance* The chemical balance of the body is far too complex a subject to be entered into here but it should be pointed out that the problem of fluid balance implies among other considerations maintenance of the acid base balance as well as the maintenance of the proper protein content of the body The concentration of electrolytes in the body cells and fluids is maintained at normal levels by maintenance of the fluid balance The relation of sodium to chloride and bicarbonate is kept constant by maintenance of the proper urinary output If one or the other of these elements is decreased in amount or is taken in excess there will be a corresponding disturbance in water balance and in turn in urinary output In short water balance is maintained only when fluid intake and fluid output are approximately equal and chemical balance is maintained only when water balance is maintained since the body fluids hold the chemical elements of the body in solution and in normal concentration

The water and salts of the blood plasma and interstitial spaces diffuse back and forth and maintain a constant equilibrium Carbon dioxide and nitrogenous end products formed in the tissues enter the blood stream by these routes excretion and production being balanced by respiratory and kidney function The pH of the blood which has a normal average range of 7.38 to 7.40 is kept within this narrow limit by preservation of the ratio between the carbonic acid (8 volumes per cent) and bicarbonate (60 volumes per cent) ion of the blood Disturbance of the balance is corrected under normal circumstances by hyperfunction so to speak of the lungs and the kidneys

*Nutritional Balance* When the protein content of the diet is insufficient over a long period of time a breakdown of the bodily store of protein occurs as evidenced by weight loss and general malnutrition Deprivation of food over a period of days vomiting diarrhea losses by way of open wounds and fecal fistulas and similar causes result in a short period of time in a high degree of protein loss as evidenced not

6000 cc the first day of treatment. After the initial dehydration is corrected the daily administration of 3500 cc of fluid or less is usually sufficient.

The scientific way to determine the water and chemical balance of the body is undoubtedly by laboratory studies but such tests however valuable they may be cannot be made daily in the laboratory of the average hospital particularly the small hospital for reasons of lack of personnel and of increased expense to the patient or if the patient is indigent to the hospital. The method suggested by Coller and his associates is adequate practical and simple. The optimum urinary output is regarded as 1500 cc and a significant decrease below that measured amount (certainly below 1000 cc) is regarded as indicative of either an insufficient daily intake of fluid or of an excessive loss of water of vaporization assuming of course that there is no renal dysfunction to explain the lessened output. In the absence of cardiovascular or renal disease a patient who is voiding 1500 cc of urine daily can be assumed to be in a state of proper fluid balance. If in addition to an optimum urinary output his mouth and tongue are moist and he suffers no subjective sensations of thirst and does not complain of dryness of the mouth it may be assumed that his fluid balance is satisfactory.

*Sodium Chloride Replacement* Coller and his associates formerly advised that a sodium chloride deficit in surgical patients be treated as follows: for each 100 mg per cent that the plasma chlorides need to be raised to reach the normal of 560 mg NaCl per 100 cc the patient should be given 0.5 gm of sodium chloride per kilogram of body weight. These observers always warned that physiologic salt solution must not be used in amounts beyond the calculated needs since it will cause fluid retention and may result in generalized edema and fatal pulmonary edema.

In 1944 Coller and his co-workers retracted the rule that for each 100 mg per cent that the plasma chloride is below 560 mg per cent 0.5 gm of salt per kilo of body weight be given. They advanced two reasons: (1) that their work indicated that oliguria accompanying salt intolerance is best relieved by the administration of sodium not in combination with chlorine or anions of other strong acids and (2) their observation of an increased tendency for salt retention in hypoproteinemic states.

Over a period of 3 months Coller and his associates reported they had observed 12 patients in whom almost disastrous results followed the application of their former rule which they now regard as highly inaccurate and dangerous. It was formulated on the assumptions that the

associated. How great the losses from tissue destruction can be is evident in studies at Army paraplegic centers which showed losses from decubitus ulcers ranging to as much as 25 gm in single patients over a 24 hour period. Army neurosurgeons also observed that the correction of malnutrition including an increased protein intake did more than any other single factor to stop the breakdown of tissue which is inherent in the existence and spread of bedsores. The patient who undergoes appendectomy also suffers a weight loss. In 23 patients submitted to this operation and hospitalized for an average of 99 days Riegel and her associates found that the average weight loss was 6.4 pounds. It could be attributed to the few hours of starvation before operation, the restricted intake afterward and the nitrogen breakdown or catabolism which commonly follows surgery. Clinical manifestations of protein deficiency include weakness, asthenia, anorexia, nutritional edema, lowered resistance to infection and impairment of liver function; a fatal outcome is not uncommon.

This very elementary statement of a very complex problem is nonetheless sufficient to make clear its essentials. Substantially it amounts to this: The normal subject in response to the normal desires of hunger and thirst provides for the maintenance of his own water and chemical balance. The sick subject either does not feel these desires, particularly the sensation of hunger, or could not gratify them by the usual channels if he did. It is therefore the responsibility of the surgeon to supply his bodily needs by abnormal channels, chiefly by way of the veins. Absorption is not usually satisfactory by the subcutaneous route and it is impractical to administer by it the large quantities of fluid which the patient needs. Proctoclisis is equally unsatisfactory. Absorption is inadequate; the patient is kept in constant discomfort and fluid is lost during bowel movements as well as by leakage about the tube. Another disadvantage is that the introduction of fluid into the rectum excites peristalsis and may throw a dangerous strain upon the appendix or the appendiceal stump.

*Principles of Replacement Therapy.* Replacement therapy is a matter of principles as well as of mathematics. On the assumption that a sick patient with definite signs of dehydration weighing 60 kg. has lost approximately 6 per cent of his body weight in fluid, he would require 3600 cc. of fluid to correct that loss. In addition he must be given enough fluid to make up the insensible loss through the skin and lungs and to provide an excess to permit a normal urinary output that is 1000 to 1500 cc., preferably the latter. An extremely dehydrated patient might occasionally require the (cautious) administration of as much as

patients with salt intolerance may be a symptom of the fluid shift since brain cells are especially sensitive to change

If intravenous infusion is indicated after operation Coller and his associates recommend in view of these observations that hypotonic solutions consisting of 0.45 per cent sodium chloride or better 0.38 per cent sodium chloride plus 0.11 per cent sodium bicarbonate should replace the isotonic solutions now commonly used

*Dextrose Therapy* A patient who cannot be fed by mouth must be fed by other methods and at present parenteral feeding with dextrose in spite of the defects to which Ravdin and his associates have called attention is still the most satisfactory method available Dextrose is not only valuable for the nutrition of the patient but is also a protection for the liver which suffers severely in the state of toxemia which accompanies peritonitis My own studies and the studies of others more over have shown that a postoperative depression of liver function occurs after all types of anesthesia is most marked for 24 to 48 hours after operation and may continue for longer or shorter periods thereafter

The administration of dextrose is related to other phases of the water and chemical balance Dehydration seriously impairs the ability of the liver to utilize dextrose while its too rapid administration on the other hand results in the loss of a considerable amount of injected sugar through the kidney glomeruli before it has been given a chance to be utilized by the liver Experimental evidence has also shown that fatty changes in the liver which occur in infection and toxemia are quite as important as the depletion of hepatic glycogen Indeed the studies of Ravdin and his associates have shown that the protective action of large stores of liver glycogen depends in great part upon the fact that the deposition of glycogen in the liver brings about a displacement of the fat content The studies of these same observers have also shown that unless there is an adequate protein supply the full use of the injected dextrose does not occur and that a high glycogen content and a low lipid level do not necessarily go hand in hand Plasma transfusion is the quickest emergency method of correcting this state

Dextrose therapy should be administered regularly and steadily to the patient who is receiving no food or little food by mouth in order to maintain him in a state of adequate nutrition a state of optimum nutrition desirable as it would be scarcely can be expected under the circumstances This form of therapy should be continued until abdominal distention has been completely controlled flatus has been passed per rectum temperature and pulse are falling to normal levels and im



chloride level of the plasma varies directly with the extracellular fluid volume and that plasma chlorides maintain a fixed relationship with the sodium ion. Both suppositions are false. The chloride level of plasma can vary without significant change in the hydration of the body and the plasma chloride level alone does not serve as a sufficiently accurate base for the construction of a universal hydration formula.

Coller and his associates now advise that the restoration of extracellular fluid deficiencies should not be made primarily on the basis of variations from the normal of ions or molecules in the blood but upon the physiologic response of the individual patient to test doses of parenteral fluids. They recommend that no salt solution or Ringers solution be given during the operative period or the immediate 48 hour postoperative period unless loss of extracellular fluid occurs as the result of diarrhea, sweating, fistulous drainage or similar causes. Otherwise glucose in 10 per cent solution is given in amounts sufficient to allay thirst completely and keep the specific gravity of the urine at least in the 1.010-1.020 range. After this period 1 liter of Ringers solution is given daily (in excess of the volume for volume replacement of drainage by 0.5 per cent Ringers solution) until the carbon dioxide combining power of the blood and the plasma chloride values are constant. If they reach a plateau before their arbitrary normal values are reached no attempt is made to change them further. Whether or not salt solution is used at all depends upon the clinical status of the patient; this is the safest plan for the salt conserving capacity of the kidneys is usually practically perfect.

In a later study Coller and his associates studied the rates of excretion of urinary constituents after the administration of salt solutions of various composition and tonicity to patients undergoing major surgical operations who were free of gross cardiovascular and kidney disease. Their conclusion was that contrary to animal experiments surgical patients cannot be assumed to have a zero salt and water load at the start of operation and there is little need for the introduction of more than 2 gm. of salt which is the compensation for the average amount of blood lost at the procedure studied (abdominoperineal resection). Even if a hypotonic salt solution is injected therefore after due allowance for what is lost in the urine and by way of the wounds there remains an excess which can only embarrass a water balance already strained by the exigencies of operation and its sequelae. One symptom of salt intolerance is edema which may result not so much from retention of water with salt as from the shifting of water from the intracellular to the extracellular spaces. The disorientation frequently observed in

glycosuria becomes negligible or disappears. Glycosuria that represents less than 10 per cent of the infused glucose is not a cause for anxiety.

Winslow's tables show that utilization of dextrose seems to proceed at the same rate regardless of the condition of the patient but it was striking and is in line with my own studies to observe that patients who had had adequate and prolonged carbohydrate therapy before operation utilized dextrose much more effectively after operation. These facts Winslow pointed out are further proof of Allen's dextrose paradox—a term used to designate that remarkable power of every non-diabetic organism to utilize dextrose in absolutely unlimited quantities—the amount of sugar utilized is governed not by any restriction of power on the part of the organism—it is governed only by the dose.

Insulin should not be used with dextrose in nondiabetic subjects. The experimental studies of Althausen made this clear and also showed that greater quantities of glycogen were deposited in the liver when insulin was omitted particularly when the initial hepatic glycogen was low. Under the latter circumstances the administration of insulin with dextrose caused liver glycogen to decrease still further. Ravdin, Brentano and others have confirmed these observations.

Mueller recommended the addition of 5 to 10 per cent alcohol (95 per cent U. S. P.) to saline or dextrose saline infusions and in 1939 reported that he had used the method 2,000 times. The normal subject can oxidize per hour 10 cc of alcohol taken orally and 100 to 150 cc is not an excessive amount to be given intravenously over a 24 hour period. The administration of the alcohol in addition to doubling and tripling the amount of easily assimilable carbohydrate supplied to the patient keeps him in a desirable state of drowsiness and euphoria so that other sedatives are needed in lesser quantities or not at all. The effect on the hopelessly ill patient is particularly fortunate—he dies in comfort. Mueller also recommended the method in the management of chronic alcoholics who require operation; several patients in his series who showed signs of developing delirium tremens and who were not controlled by sedation were promptly controlled by the use of intravenous alcohol.

According to Mueller injections of solutions containing alcohol can be given for 48 to 72 hours without sclerosing effects. Schullinger, who recommended this form of therapy for paralytic ileus, stated that local venous thrombosis usually follows its employment.

Moore and Karp also recommended the intravenous administration of alcohol. Their preference is to add 50 cc of 95 per cent alcohol to 1,000 cc of 5 per cent dextrose solution the total intake thus being 260

provement is clear from every other aspect. The principle of the treatment of peritonitis is physiologic rest and that principle is violated if gastro-intestinal activity is permitted by the ingestion of food. Intravenous alimentation must therefore be relied upon until oral feeding can safely be resumed. An additional reason for the regular administration of dextrose is to obviate periods in which the hepatic glycogen would be reduced to dangerously low levels or to levels that would require the later administration of increased quantities of dextrose before restoration to normal could occur. Bower lays particular emphasis upon this point.

Winslow's controlled studies on the utilization of glucose in surgical patients showed that when 5 per cent dextrose solution was used 98 per cent was utilized and when 10 per cent solution was used 95 per cent was utilized. He therefore concluded that 5 per cent is best for routine use because it is isotonic for the blood, the content is sufficient to prevent ketosis and provide ideal food for energy, the liver is adequately protected and edema is avoided since physiologic salt solution is used only as indicated. The administration of 3 liters of dextrose solution per day at rates of 300 to 500 cc per hour is sufficient for patients in fair general nutrition who will shortly be restored to oral feeding but the amounts may be increased as necessary. About 98 per cent of the sugar which averages 0.35 gm per kilo of body weight each hour is utilized.

Ten per cent dextrose solution is recommended when the demands by the organism are greater. A solution of this strength is hypertonic with blood, is mildly diuretic and when administered at 300 to 500 cc per hour provides 93 per cent more carbohydrate than an equal quantity of 5 per cent solution. It is preferably administered at the rate of 200-300 cc per hour. Generally speaking the more fluid is likely to be needed the slower should be the rate of administration.

Recent studies of parenteral glucose feeding by Prentiss and Somogyi indicate that glucose is most efficiently administered in solutions of 10 per cent concentration begun at a slow rate (10 to 12.5 gm) for the first 30 minutes then accelerated to 50 to 60 gm per hour until the infusion is completed. Urine specimens obtained during and for several hours following infusions are quantitatively analyzed for sugar content by the method described by Somogyi. Serial quantitative urinary sugar determinations are made in patients who show more than minimal glycosuria or who are suspected of markedly depressed glucose tolerance. In these cases the rate of infusion is not accelerated until

0.30 gm of nitrogen per kilogram of body weight per day and an intake of at least 30 calories per kilogram of body weight per day are desirable during the postoperative period if the nitrogen balance is to be maintained. Elman has pointed out that the necessary intake to prevent a negative nitrogen balance is 62.5 gm of protein (10 gm of nitrogen) daily. If protein depletion is severe several times that amount is desirable and may be lifesaving; it should be given in combination with glucose which is protein sparing. Total intravenous alimentation over periods of time from 10 to 55 days has been reported as Bigham and his associates noted in a report of their own experiences.

Whenever possible the food intake should be by mouth. Concentrated protein food such as dried whole milk, soybean powder, dried brewers' yeast or one of the powdered protein concentrates or hydrolysates can be added to the diet without increasing the bulk. The protein intake can thus be brought up to 250 or 300 gm per day and with care and ingenuity the feedings can be made palatable and attractive.

In a patient who cannot take protein by the most desirable route that is by mouth the use of amino acids which represent the basic nutritional elements of all protein is an acceptable and practical substitute. They are available commercially in a safe (Council accepted) preparation and their use is practically without risk if they are properly administered and if the proper contraindications (such as severe liver damage and cardiac and renal insufficiency) are observed.

Theoretically as Elman pointed out it would be desirable to correct chronic protein deficits promptly and from the theoretical standpoint there is no reason why this should not be done. Practically however this would require the administration of very large doses the safety of which has not yet been ascertained. The administration of 2 to 3 liters daily of a 5 per cent solution of amigen<sup>1</sup> in 5 per cent glucose solution supplemented by whole blood transfusions or an occasional plasma transfusion is safe and is adequate for the ordinary patient. Two liters of amigen contain 100 gm of protein, 100 gm of carbohydrate and 5 gm of sodium chloride which will meet the minimum daily nutritional requirements of the average patient. The solution should be given slowly. When amino acids are given by vein great care is necessary to prevent contamination as the solution is an excellent culture medium.

In patients who are able to take food via the intestinal tract tube feeding may be resorted to. Special preparations have been devised containing skimmed milk, skimmed milk powder, liver extract, brewers' yeast and similar items together with vitamins but the average patient does not tolerate tube feeding well and may vomit if it is resorted to.

calories instead of the 200 calories available when only dextrose is used. They also noted occasional sclerosis of the vessels in which the injection was made. In 1 case partial ulnar nerve palsy was possibly caused by the presence of subcutaneous alcohol in the cubital fossa.

**Transfusion** Peritonitis that follows rupture of the appendix and that continues for a long period of time almost invariably results in anemia for which transfusions of whole blood should be given. Transfusions may also be necessary to correct the destruction of red blood cells which may result from even careful use of the sulfonamide drugs. Wright and his associates added two other indications for transfusion: (1) a mounting pulse and falling blood pressure to be interpreted as evidence of impending shock, more and more blood stagnating in the splanchnic and mesenteric systems as less and less is fed to the heart for the peripheral circulation; and (2) a marked fall of the total white blood cell count which shows that the hemopoietic system is being overcome by infection and its products. The assumption is that function is temporarily suppressed and that the bone marrow is not primarily damaged since it responds to transfusion by renewed activity.

In a study by Hardy and Godfrey of the effect of intravenous fluids on cardiac output, stroke volume, pulse rate and blood pressure of 5 dehydrated patients as compared with 6 control patients, the dehydrated group manifested a prompt and significant rise in cardiac output manifested chiefly by an increase in stroke volume accompanied by a small increase in pulse pressure. The writers therefore concluded that the feeling of general well-being so often experienced by dehydrated subjects after intravenous fluid administration is accompanied by concomitant objective changes in circulatory dynamics. That the maximum effect is often produced by the administration of the first liter suggests that this amount may be sufficient in patients in whom too much intravenous fluid would be dangerous.

**Protein Replacement** When the importance of protein depletion in surgical patients first began to be appreciated, it was thought that plasma transfusions were the best method of replacing the deficit. It is now known that this method in addition to being expensive is not efficient except in acute emergencies. Plasma furthermore contains a large amount of sodium in the form of the sodium chloride normally in the blood as well as that added in the form of citrate to prevent clotting and the amounts in which it would have to be given to secure an adequate amount of protein would place an intolerable burden on the circulation.

Riegel and her associates have pointed out that an intake of at least

serious degree of anoxia. Anoxemia is also associated with the toxemia which always accompanies peritonitis. The rationale of oxygen therapy is therefore so obvious that it needs no special discussion.

The good results produced by oxygen inhalations are numerous. Its routine use materially reduces the incidence of postoperative pulmonary complications (p. 245). It is valuable in the prevention and treatment of surgical shock. Oxygen prevents or decreases the liver damage caused by anoxemia and is particularly valuable when there is pre-existing liver damage (Judd, Snell and Hoerner, Ravidin *et al.*). It also prevents or decreases the cerebral damage associated with anoxemia.

Finally the observations of Fine and his associates have shown that the use of oxygen promotes the absorption of gas from the distended intestine and is particularly helpful in combination with constant intestinal decompression. Putting the explanation into the simplest terms, the gas in the distended intestine consists largely of swallowed air and its nitrogen content is therefore high. The inhalation of pure oxygen reduces the partial pressure of nitrogen in the alveoli. As a result there is a decrease of the nitrogen in the blood. This decrease results in an increase in the gradient of diffusion into the blood stream of the nitrogen contained in the distended bowel. Finally, the dissolved gas (i. e. nitrogen) is carried out through the lungs.

The decompressing action of oxygen inhalations is therefore due not to any properties in the oxygen itself but to the fact that as nitrogen is shut out of the inspired air the direction of flow of nitrogen must be away from the intestine and toward the lung and that diffusion into the intestine is thus prevented. When oxygen was substituted for air in experimental observations on obstructed cats a decrease of 26 per cent in the total gas volume of the small intestine was observed after 8 hours and 61 per cent of the total volume had been absorbed within 18 to 24 hours. The efficacy of oxygen therapy used for this purpose is directly proportionate to the concentration in which it is used.

Oxygen is of greatest value if it is used before its necessity is evident. A patient suffering from oxygen lack has a high pulse rate, shallow rapid respirations and a grayish or actually cyanotic hue. Patients treated routinely by oxygen after operation for advanced appendiceal disease or treated by it as a routine part of the conservative treatment of peritonitis are not likely to exhibit these manifestations and will be in correspondingly better state.

Oxygen is most conveniently administered by intranasal catheter, the Boothby-Lovelace mask or some similar device and its use has been greatly simplified by the introduction of these methods. If the

**Vitamin Therapy** Patients who are vomiting and who are unable to take food by mouth over prolonged periods of time will obviously develop vitamin deficiencies which should be treated by the administration of vitamin preparations according to the indications. A thiamine deficiency may be responsible for anorexia which in turn is responsible for malnutrition and protein deficiency. The parenteral administration of thiamine hydrochloride is not entirely free from risk. Ringold and Webb in reporting a personally observed fatality apparently due to anaphylactic shock mentioned a previous death reported by Mills after the intravenous administration of thiamine. Their suggestion is that oral administration be employed whenever possible and that the vitamin preparation be tested as an allergen if parenteral therapy must be used.

As a general rule the daily administration of 1 gm. of ascorbic acid, 200 mg. of nicotin, 20 mg. of riboflavin and 20 mg. of thiamine answers all vitamin requirements.

**Replacement Therapy in Patients with Cardiovascular and Renal Disease** Patients with cardiac or renal disease present a special problem in the maintenance of fluid balance. They must be treated in co-operation with a competent internist and laboratory studies and mathematical calculations are necessary to determine the amounts of sodium chloride they should receive. If a tendency to edema is evident sodium chloride should be restricted or withheld entirely. The circulation of these patients is often impaired and it may be necessary for this reason to give them less fluid than would otherwise be desirable. Studies of the urea and nonprotein nitrogen of the blood should be made daily.

**Termination of Replacement Therapy** Parenteral therapy should be discontinued and oral feeding resumed at the earliest possible moment. This is an emergency method of the greatest usefulness in an emergency but not to be continued beyond it. Hardin and his associates have properly warned that the tendency to place more and more emphasis on the value of intravenously injected glucose is fraught with the risk of overlooking the simpler administration of food by mouth. Althusen's demonstration which I have corroborated of the greater value as well as the greater simplicity of administering dextrose by mouth is in accordance with these observations.

#### OXYGEN THERAPY

The reduced vital capacity observed after all operative procedures on the abdominal organs is increased in peritonitis by the intestinal distention which frequently accompanies it and which may result in a

Johnson in discussing Morginson's report of reactions to penicillin stated that in Army hospitals his experience was that casualties who had not previously been treated with penicillin showed a reaction rate of not more than 2 or 3 per cent. In private practice as the general population was becoming sensitized by the use of penicillin for minor conditions the same situation was developing as had already come to pass with the sulfonamide drugs and he himself was observing a reaction rate of 30 per cent in chronic infections and eczematoid dermatoses.

3 Neither the sulfonamides nor penicillin should be used to accomplish anything which can be better accomplished by surgical measures. Pus still remains a surgical lesion. The entire military experience pointed to the fact that all such agents are useful only in proportion to the proper use of surgical measures.

4 Chemotherapy is not a panacea in peritonitis but merely one phase of treatment.

5 Although the reduction in the morbidity and mortality of appendiceal peritonitis coincided with the introduction and wide use of chemotherapeutic and antibiotic agents it also coincided with the general use of such measures as plasma and whole blood transfusions, other replacement therapy on a rational basis, the prevention of hypoproteinemia, the almost routine use of intestinal decompression, the administration of oxygen in high concentrations and similar measures.

6 Chemotherapeutic and antibiotic agents are of limited usefulness in the treatment of localized appendiceal abscesses, whether primary or secondary, or of gangrene of the appendix. Their failure in such conditions is reasonably explained by their inability to gain access to the organisms because of the inadequate circulation in dead tissue and walled off tissue. It may also be, as Jackson and Collier pointed out in connection with the sulfonamides, that an exudate rich in products of tissue necrosis contains substances that inhibit the usefulness of such agents.

*Routine of Administration.* The literature concerning the sulfonamides and penicillin is now so extensive and so detailed that there is no justification for repeating here very much concerning these agents. At the present time the preferable method of administration is as follows:

1 Penicillin is given intramuscularly in dosages of at least 50,000 units every 2 or 3 hours for at least 5 days. If improvement is evident by this time 30,000 units are given every 3 hours for another 4 days. If this plan or some equivalent plan is not employed or if massive



intranasal catheter is used a continuous flow of 5 to 8 liters provides a concentration of 40 to 60 per cent in the inspired air. If concentrations of 60 per cent and over are desired the oxygen mask provides high concentrations with a relatively low rate of flow. Concentrations of 60 per cent are most generally used for all purposes but concentrations of 95 per cent are used for the treatment of intestinal distention and Boothby, Mayo and Lovchick who advocate concentrations of 90 to 100 per cent for shock have seen no ill effects although they believe that administration of oxygen at this rate of concentration should not usually be continued for more than 48 hours.

In addition to convenience of administration the introduction of the oxygen mask and the intranasal method of administration have reduced the cost of oxygen therapy to a level that makes its use practical for most patients even in public institutions. The elimination of the oxygen tent has also had a good psychologic effect since many patients were frightened by the elaborate apparatus formerly necessary when oxygen was used.

#### CHEMOTHERAPY AND ANTIBIOTIC THERAPY

In view of the excellent results achieved in various infections by the use of sulfanilamide and its derivatives it was logical to employ these drugs in the treatment of advanced acute appendicitis. In the first flush of enthusiasm for them and later for penicillin (although when it was introduced evaluation was far more judicious) it might have been assumed that the problem of appendiceal peritonitis had been completely solved. At the present time however the following concepts are generally held:

- 1 The introduction of chemotherapeutic and antibiotic agents has not in any way solved the problem of acute appendicitis complicated by peritonitis and abscess. The solution of that problem is still prophylactic: the prompt removal of the unruptured appendix.

- 2 However effective they may be the sulfonamide drugs are potentially dangerous agents and their promiscuous use is unwarranted. Penicillin is considerably less toxic though it may give rise to reactions. The long continued unwise use of both drugs may result in the development of resistant strains of micro organisms and the possibility also exists that because of the rather general tendency to use drugs of such potency for trivial ailments patients may be sensitized against them so that their use in major conditions will be less effective and perhaps entirely impossible.

as the diagnosis is established an effective blood level can be secured promptly.

Griffin and his associates recommended the prophylactic use of penicillin and sulfadiazine in all cases of acute appendicitis. Their preliminary report included 62 cases of acute appendicitis with suppuration, 17 cases with gangrene and 29 cases with perforation. Penicillin in the amount of 20,000 units is given as part of the preoperative preparation and is continued in the same dosage every 3 hours after operation until a total of 660,000 units has been given. Sulfadiazine in 1 gm doses is given 4 times daily for 4 days. If the intravenous route must be used the dosage of the sulfonamide is proportionately reduced. The method seems therapeutic rather than prophylactic in complicated acute appendicitis and one doubts the justification for the trouble, expense and possible danger of reactions in uncomplicated cases.

Chemotherapy as a substitute for surgery was employed in 8 cases diagnosed as acute appendicitis by Berkeley and Watkins aboard a Naval transport operating under wartime conditions on the theory that this method was more desirable than surgery under unfavorable circumstances. The patients were admitted within 2 to 6 hours after the onset of symptoms. 7 were treated with sulfathiazole (2 gm as the initial dose followed by maintenance doses of 1 gm 4 times a day) and 1 was treated with sulfanilamide by the same routine. All showed progressive diminution of pain, tenderness and rigidity after the first 12 hours, a progressive decrease of the white blood cell count and complete disappearance of subjective symptoms within 24 hours. Residual tenderness persisted for 2 to 4 days. The writers were of the opinion that chemotherapy was the apparent answer to the management of acute appendicitis under conditions unfavorable for surgery by changing acute appendicitis from a surgical emergency to a condition of simple medication. They continue:

Our incomplete studies even suggest that except in the most unusual circumstances chemotherapy might entirely supplant surgery in the treatment of acute appendicitis when operating conditions are unfavorable. Many cases will promptly clear under this management but should surgical intervention become advisable little additional risk to the patient will have been incurred while maintaining a satisfactory blood concentration of sulfonamide. Such additional risk as is incurred we believe is far outweighed by the great majority of cases that promptly clear without the hazards of operation.

One hesitates to take issue with medical officers who have experienced combat conditions but on the surface this seems a dangerous form of treatment.

dosages are abruptly discontinued a recurrence of symptoms and the appearance of masses (assumed to represent abscesses) are frequent.

2 Although experimental evidence is contradictory as to the value of penicillin and a sulfonamide drug used simultaneously, the clinical evidence suggests that combined treatment is of value. The work of Bigger indicates that sulfathiazole is more effective than either sulfanilamide or sulfapyridine in combination with penicillin. When therefore penicillin alone in adequate dosages does not seem to be producing the desired effects, the addition of sulfathiazole is often useful. The initial dose in the usual case should be 2 gm. and the maintenance dose 1 gm. 4 to 6 times daily by mouth if the patient can be given medication by this route. If parenteral administration is necessary, sulfadiazine is preferred to sulfathiazole because of its slower excretion. It is used in the form of the sodium salt in 5 per cent solution in distilled water or physiologic salt solution. A concentration of 6 to 8 mg. per cent in the blood is desirable though if the patient is critically ill 10 to 15 mg. per cent is better. These are general observations. Actually the dosages of both penicillin and the sulfonamides must be regulated by the necessities of and the response in the individual case.

Penicillin is ordinarily used as the sodium salt which is readily soluble in isotonic solution of sodium chloride and in 5 per cent dextrose solution. It is important that the drug be begun promptly and be administered in large doses. Gauley and his associates who produced fulminating diffuse peritonitis in dogs by ligation of the base of the appendix and the mesentery and blood supply, had no deaths from peritonitis (except in cases complicated by fecal fistula) when penicillin therapy was begun within an hour after operation but had a 21 per cent mortality when administration was delayed for 12 hours. The mortality in their control animals was 92.6 per cent. On the basis of dosages in experimental animals these observers concluded that the dosage in man should be at least 22,500 units per hour, or 500,000 units daily. Crile who used penicillin therapy in 25 patients with spreading and 25 patients with localizing appendiceal peritonitis, all of whom were very ill, used dosages of 100,000 units every 2 hours for at least 11 days.

Beecher's suggestion for wartime casualties with peritonitis might perhaps be applied in patients with appendiceal peritonitis. He pointed out that an average of 2 hours is spent in the examination and resuscitation of the patient and that anesthesia and operation account for another hour. If the sulfonamide drug (or penicillin) is begun as soon

*Local Chemotherapy* Sulfonamide preparations seem first to have been employed in the peritoneal cavity in perforated appendicitis by Dees and to have been popularized by Thompson and his associates at the Roosevelt Hospital in New York. Even when the greatest enthusiasm for this method was being reported its undesirable features were appreciated the chief being that absorption could not be controlled which made the intraperitoneal use of such preparations theoretically at least the most dangerous method of use. The sulfonamides are being used less and less by this method for the basic reason that just as much protection and possibly more protection is secured by systemic administration without the dangers inherent in introducing a foreign material into the peritoneal cavity.

On the other hand some few authorities still continue enthusiastic about the method. Mueller in 1945 reported that sulfanilamide had been used in the peritoneal cavity in 320 out of 739 cases of acute appendicitis and its complications over a 5 year period at the Roosevelt Hospital he attributed the mortality which had been reduced to 0.4 per cent (from 2.83 per cent over the comparable previous 5 year period) and the reduced number of complications to this method. penicillin was used in abscesses and peritonitis in addition to local sulfanilamide and those who question the use of local chemotherapy might be inclined to attribute the improved results to penicillin.

Young and Cole reported in 1946 that the experimental intraperitoneal administration of succinylsulfathiazole and phthalylsulfathiazole was effective and harmless in therapeutic doses and gave rise to no foreign body reactions. Both agents were then used clinically by this route with good results. The argument again arises however that if as good results can be secured without the intraperitoneal use of the sulfonamides as with it as they apparently can be there seems little point to resorting to it.

If the surgeon considers intraperitoneal administration desirable for special reasons in a special case the studies of Throckmorton suggest that sulfathiazole is the most desirable agent since it is specific against a large number of micro-organisms it has a prolonged bacteriostatic effect it seems to be effective as a peritoneal vaccine and its undesirable local effects are minimal. If this method is employed the surgeon should remember that toxic reactions may occur rapidly that jaundice is particularly frequent and that the sulfonamides should be omitted altogether by the systemic route or should be withheld until 18 hours or more after their intraperitoneal administration.

*Reactions* Sulfonamide drugs are not free from risk and it is un-

Crick's summary of the response of peritonitis of appendiceal origin to penicillin in massive doses is typical of the usual successful response

For 18 hours there is little or no change in the general condition of the patient but peritonitis does not seem to spread

On the third day the clinical condition is improved. The pulse and temperature are lower the abdomen is less rigid and tender ileus subsides and the patient feels better

By the fifth or sixth day the patient feels well takes soft diet and is alert and comfortable

By the seventh day although intra abdominal masses may still be present there are no clinical symptoms and the temperature is low

If improvement is not definitely evident by the fourth day mechanical obstruction of the small intestine should be excluded by roentgenologic examination. If between the eighth and the twelfth days after penicillin has been discontinued the patient complains of abdominal pain or tenderness and gas pains and if fever is more than minimal recrudescence of infection in abdominal abscesses should be suspected and a second course of penicillin should be given

The results secured by penicillin therapy in peritonitis of appendiceal origin can be explained according to Crick by the action of this antibiotic against the virulent gram positive cocci present *Escherichia coli* and other penicillin resistant organisms are almost always found in cultures taken from patients with appendiceal peritonitis and penicillin in amounts up to 20 units per cubic centimeter was shown by Altmeier not to inhibit the growth of *Esch. coli* according to these studies if a known amount of penicillin is incubated with a culture of *Esch. coli* 98 per cent of the activity of the penicillin is destroyed in 12 hours. This microorganism in pure culture however is usually of low virulence and it is reasonable to assume that if virulent gram positive cocci present in appendiceal peritonitis can be controlled by antibiotic therapy the peritonium will be able to localize and eventually to absorb infections due to *Esch. coli*. This reasoning explains why massive doses of penicillin are required to control gram positive cocci which are sensitive to penicillin when they are present in a mixed infection and are associated with *Esch. coli* and why several days are required to secure results. By giving sufficient penicillin over a sufficiently long period of time a stage is finally reached at which more penicillin is given than *Esch. coli* can destroy and the concentration of active penicillin is therefore sufficient to inhibit the growth of virulent cocci. Crick's own experimental studies with staphylococci in pure culture and in mixed culture with *Esch. coli* confirmed his reasoning

chemotherapeutic agent presently available which presents such a wide margin between therapeutic effectiveness and toxicity. None of the complications which it causes are lethal and most of the time it gives rise to no reactions at all.

**Streptomycin** Streptomycin, an antibiotic substance formed by a certain strain of the fungus *Actinomyces griseus*, has been extensively studied experimentally and has been used under controlled conditions in Army hospitals and in research projects under the direction of the National Research Council but has only recently become available commercially.

Murphy, Ravdin and their associates in an experimental study found streptomycin in significant amounts in the peritoneal fluid following its systemic administration. The survival rate for streptomycin-treated animals with peritonitis was 60 per cent against 30 per cent for control animals. Studies on normal animals also showed appreciable amounts in the peritoneal fluid. When dosages of 75 and 90 mg. were given every 4 hours, fairly high concentrations could be secured in both blood and peritoneal fluid even when the dosage was later decreased.

Silvani and his associates in an experimental study of fulminating diffuse peritonitis of appendiceal origin found that streptomycin used systemically and locally prolonged life in the dosages used but did not significantly alter the bacterial flora of the peritoneal exudate nor was it lifesaving. Dosages used intraperitoneally which sterilized the peritoneal fluid invariably killed the animals by respiratory failure apparently of central origin.

Clinical reports to date are few. The Committee which studied streptomycin for the National Research Council reported 21 cases of appendicular peritonitis with 18 recoveries. All patients had 1.2 gm. daily by the intramuscular route but it was concluded that in these circumstances at least 2 gm. per day should be given for 5 days or longer. This is equivalent to 2,000,000 units, a unit being defined as the antibacterial activity of 1 microgram of pure crystalline streptomycin base.

Hirshfeld and his associates used streptomycin in 12 cases of peritonitis but were not greatly impressed with the results. In 8 of these cases penicillin and the sulfonamides were also used.

Pulaski, Seeley and Matthews recorded the Army experience with 39 streptomycin-treated patients with peritonitis of appendiceal origin which in 17 instances was of the spreading and in 22 instances of the localized variety. There was 1 death in each group due respectively to pulmonary infarction and to subarachnoid hemorrhage. Results were

fortunate that the impression has spread that they are so effective that their side effects may be disregarded. They must be used under very careful observation and both nursing and medical staffs must clearly understand that no deviation from the normal is too slight to be evaluated seriously.

The literature on this subject is full and requires no lengthy resume here. All of the sulfonamides have a deleterious action on the red blood cells and for this reason blood counts should be made at regular intervals when they are used. Mild degrees of anemia can be treated by iron, liver extract and similar measures. If an extreme degree develops the drug must be discontinued at once and repeated transfusions given. Cyanosis is not an indication *per se* for discontinuing sulfonamide therapy but is definitely an indication for redoubled observation and precautions.

Nausea and vomiting caused by these drugs are difficult to distinguish from similar symptoms caused by the primary disease. The same is true of fever though it frequently begins about the time one would expect fever from the primary disease to disappear. If the temperature elevation is not believed to be due to the primary disease the drug must be discontinued at once.

Renal complications include hematuria, oliguria, impairment of renal function and terminal uremia. Fatal cases have been reported and if hematuria or oliguria develops sulfonamide therapy must be discontinued at once.

Juandice is a relatively infrequent but an extremely serious complication of sulfonamide therapy since it indicates hepatic injury. The drug should be promptly discontinued if it occurs.

Penicillin has a very low toxicity and reactions during its use are infrequent. Pain at the site of the injection occasionally occurs but is insignificant. Since the pure substance has become available chills and fever are infrequent. Urticaria may follow either local or systemic administration and may appear early or late in the course of treatment or even after treatment has been discontinued. It is seldom of importance. If it is severe it can be controlled by the administration of epinephrine. Miscellaneous reactions include mild abdominal pain, headache, muscular aches and pruritus and similar symptoms which are practically always of no consequence. There is no evidence whatsoever that the drug has a deleterious effect on the erythrocytes and leukocytes of the circulating blood. This is in marked contrast to the sulfonamides and makes the administration of penicillin safe regardless of the blood picture. In short as numerous observers have pointed out there is no

its use and to justify its employment only in conditions in which the safer and less toxic penicillin has proved ineffective.

#### THE ROUTINE OF EXPECTANT (CONSERVATIVE) TREATMENT IN COMPLICATED ACUTE APPENDICITIS

Whether conservative treatment is employed for spreading peritonitis following rupture of the appendix or for an appendiceal abscess the patient must be in the hospital and must be visited by the surgeon at least twice daily and preferably oftener. Competent nursing service must be available which incidentally is another argument against the general use of the method. The sort of nursing which it requires is not available to many private patients or to most patients in public institutions. The quiet private room specified by Mayo is equally desirable and as generally unattainable.

A detailed chart should be kept for comparative purposes. The temperature is taken by rectum if necessary every 2 or 3 hours. The pulse is taken at least every 2 hours. The fluid intake and output are measured and their sources recorded. Full details of the clinical progress are also recorded. If a mass is palpable it is outlined on the abdominal wall and diagrammed on the chart and daily examinations and notations are made. If the mass is in the pelvis rectal examination is carried out at least every second day.

Expectant treatment requires that food and fluids be strictly withheld with the exception that water may be drunk if the patient desires since constant decompression of the intestinal tract is part of the treatment the fluid is immediately aspirated from the stomach and does no harm. The fluid chemical and nutritional balance is preserved by the calculated use of intravenous infusions of physiologic saline and glucose solutions. Transfusions of whole blood or plasma are employed according to the indications. Oxygen therapy is employed routinely as are sulfonamide and antibiotic therapy and such other measures as the individual surgeon may prefer to use. These measures have already been discussed in full. It will be observed that the routine of conservative therapy is essentially the same as the routine employed in the management of complicated acute appendicitis treated by immediate removal of the appendix.

It is impossible to state in terms of days criteria in regard to the success or failure of expectant treatment though usually if improvement occurs at all it is evident within 48 hours and sometimes within 24 hours of the institution of the measures outlined. The danger point



good and the response was prompt in 4 cases of spreading peritonitis treated only by streptomycin and in 12 cases treated by streptomycin and penicillin sometimes with sulfadiazine also. Results were also good and the response was also prompt in 10 of the localizing cases 2 of which were treated only by streptomycin. In at least 1 case in which a clinical diagnosis of pyelophlebitis was made following appendiceal abscess treated by appendectomy and drainage the drug was regarded as lifesaving.

The authors however looked upon their results with considerable caution their conclusions being about as follows. Clear cut results were generally more apparent in spreading than in localized processes. Results were generally poor when palpable masses were present. Results were seldom dramatic whether streptomycin was used alone or in combination with penicillin though in both spreading and localized peritonitis recovery was thought to be smoother and prompter when the drugs were used in combination. The addition of sulfadiazine was not helpful. The resolution of established peritoneal suppuration was of approximately the same pattern in the patients treated only by streptomycin as in patients receiving penicillin in excess of 800 000 units. Consistently good results were never obtained with streptomycin when the dosage was less than 2 gm a day whether the drug was given alone with sulfadiazine or with penicillin in amounts of 240 000 units per day or less. The most satisfactory results were obtained when streptomycin was used alone in 3 gm dosages or in 2.5 gm dosages in combination with an average dosage of 180 000 units of penicillin. Streptomycin should probably be reserved in the treatment of peritonitis of appendiceal origin for patients who fail to respond to penicillin either alone or in combination with a sulfonamide.

Numerous reactions have been reported following the use of streptomycin most of which are of minor consequence. The only serious reaction reported with any frequency is a vestibular disturbance causing dizziness tinnitus and occasionally transient deafness or vertigo. It usually requires termination of therapy. This reaction like the aplastic anemia reported in 2 instances by Deyke and Wallace seems to occur only when large doses of streptomycin are administered over relatively long periods of time which is not usually necessary in peritonitis of appendiceal origin. On the other hand the fatal case of toxic encephalopathy reported by Hunicutt and his associates occurred on the fifth day of therapy. The experience to date thus suggests that streptomycin is of sufficient potential seriousness to warrant extreme caution in

therapy in acute appendicitis from Weinberg's first note on the subject to date

**Vaccination** The whole subject of vaccination in peritonitis was reviewed by Harvey and Meleney who did not regard the possibilities of the method as proved or disproved either experimentally or clinically chiefly because experimental conditions were variable and clinical controls were not adequate. It is doubtful that there is a field for vaccination in the management of peritonitis at the present time because of the efficacy of other methods.

**Ultraviolet Therapy** Havlicek (cited by Harvey and Meleney) in 1932 reported the treatment of 108 cases of appendiceal peritonitis without a death by the use of ultraviolet therapy following operation while the abdomen was still open. Variable results were reported by others who used this method and the rationale is not clear.

**X-ray Therapy** The chief proponents of radiotherapy in peritonitis are Kelly and Dowell who reported good results by the use of small doses at frequent intervals (2-3 times daily) at the bedside in early spreading peritonitis in fibropurulent peritonitis and in localized abscesses after drainage. The method is not recommended for early acute appendicitis or for generalized peritonitis with adhesions and abscess formation. It has not found general favor.

**Adrenal Cortex Therapy** Patients with spreading peritonitis have been shown to have extensive adrenocortical degeneration at autopsy and on that basis it has been suggested that extracts of adrenal cortex be used both as substitution therapy and as an aid to the maintenance of the electrolyte balance. The extract is added to intravenous solutions when a rising hematocrit shows that fluid administered is not being utilized. The method is not of general usefulness and its value is doubtful.

**Conclusion** The majority of these reports concern adjuvant measures which should never under any circumstances be used in lieu of early surgery and adequate replacement therapy. If they are employed with the proper precautions there would seem no particular objection to their tentative trial in appropriate cases though there also seems little field for them in view of more effective measures especially chemotherapeutic and antibiotic measures presently available.

#### PSYCHOGENIC FACTORS IN CONVALESCENCE

The personality of the patient has considerable to do with his convalescence and sometimes with the actual outcome of the case. A patient

in one patient is past in a matter of hours but in another not for days. Pain decreases tenderness and rigidity decrease with it distention is less dehydration is overcome vomiting ceases the pulse rate falls and the temperature slowly drops to normal. As Grey Turner emphasized however it should not be concluded that natural recovery is taking place unless *all* signs and symptoms improve. Those who favor the tentative plan of expectant treatment believe that operation should be performed promptly if abdominal symptoms and signs do not improve if the mass becomes larger or does not decrease in size within 4 or 5 days if pain increases if the pulse continues rapid or becomes faster and if the temperature continues high or rises.

#### MISCELLANEOUS THERAPEUTIC METHODS

**Serotherapy** At various times attempts have been made to treat appendiceal peritonitis by serotherapy but experimental evidence is meager and clinical evidence not entirely convincing. Most of the sera used have been directed against *Cl welchii* infection and their use would seem to imply a belief not warranted by the facts in the pre dominance of this kind of infection.

The most recent report on the subject by Bartels and Manicus Hansen from the Viborg Hospital in Denmark is a comparison of two decennial periods 1921-30 when serum was not used and 1931-40 when it was available. The number of cases of peritonitis within the two periods was substantially the same. In the first period there were 490 operations with 48 deaths. In the second there were 902 operations with 28 deaths which is a reduction of 42 per cent.

Adults generally received 20 to 25 cc of antigas gangrene serum and 25 cc of anticol serum children correspondingly smaller doses. The serum was injected intravenously at the conclusion of the operation and larger doses were given in severe cases. Injections were repeated during the first few days after operation. The serum was used not only in peritonitis but in gangrene of the appendix and in all cases in which the disease was of more than 48 hours duration. During 1941-3 275 surgical cases 40 with peritonitis were treated by operation serum and sulfathiazole the latter agent providing protection against streptococci against which the serum contains no component. Serum sickness was usually slight but anaphylactic shock occurred in 2 cases and in 1 was severe.

This report contains an excellent review of the literature of sero

[Beham Hillel and Perr Herbert Stomatitis due to streptomycin *J A M A* 138 495 6 (16 Oct) 1948] and Gundrum paralysis of the left vocal cord [Gundrum Lawrence K Paralysis of left vocal cord following streptomycin therapy *J A M A* 138 22 (4 Sept) 1948] A death from aplastic anemia has also been reported [Correlli F Anemia aplastica acuta mortale dopo streptomicina contributo alla conoscenza delle reazioni ematiche da streptomicina *Polichinico (sez prat)* 54 1088 1947 Abstracted in *Internat Abstr Surg in Surg Gynec & Obst* 86 610 (June) 1948]

Several cases of dermatitis have been reported in nursing personnel who prepared and handled streptomycin [Crofton John and Foreman H M Streptomycin dermatitis in nurses *Brit M J* 2 71 2 (10 July) 1948 Shapiro Seymour I and Carney Robert G Contact dermatitis due to streptomycin *J Iowa M Soc* 38 204 11 (May) 1948]

In their latest contribution on streptomycin therapy in peritonitis Pulaski and his associates (Pulaski E J Voorhees Arthur B and Seeley Sam F Further experiences with streptomycin therapy in peritonitis in press) conclude that in early spreading peritonitis of appendiceal origin combined treatment with streptomycin and penicillin seems to offer no significant advantage over streptomycin alone though combined therapy seems superior in the management of localizing lesions The optimal course of streptomycin therapy is thought to be 7 to 10 days and on empiric grounds the optimal dosage seems to be at least 2.5 gm daily Administration for at least 48 to 72 hours after the temperature returns to normal is recommended to prevent the development of an escape phenomenon The series of cases is not yet sufficiently large to permit statistical conclusions

A series of studies by Lehr culminating in 1947 [Lehr David Low toxicity of sulfonamide mixtures II Combinations of sulfathiazole sulfadiazine and sulfamerazine *Proc Soc Exper Biol & Med* 64 393 401 (Apr) 1947] show that a mixture containing partial dosages of the various sulfonamides permits a significantly greater margin of safety than any single sulfonamide in full therapeutic dosage and returns in addition the full chemotherapeutic potential of the combined factors The clinical toxicity of the mixture is unusually low as compared with that following treatment with single sulfonamides and the possibility of concretum formation in the urinary tract at the usual dosage level of 6 gm seems almost completely eliminated the low toxicity of the mixture is explained by the prevention of renal obstruction Mixtures of sulfonamides are more completely absorbed and excreted than equal amounts of the individual constituents and blood levels are distinctly

with neurotic tendencies is difficult to manage though more often ignorance and apprehension explain the state of mind in which the surgeon and his assistants must strive against mental and nervous as well as physical factors. An explanation to the patient before operation of what is to be done and if he is able to comprehend an explanation after operation of the measures to be employed will go far to combat such a state of mind. The current practice of early ambulation has as one of its chief advantages improvement in morale. Patients react to trauma, physical pain and other factors associated with illness and operation in different ways. Underlying psychiatric tendencies may come to the fore under the stress of the emergency and play an important part in the outcome. Sometimes psychologic and psychiatric investigation is actually needed. On the other hand it is quite possible to overemphasize such considerations; the modern trend is in that direction and the surgeon while paying due heed to these factors must be warned against paying too much attention to them.

In this connection it might be mentioned that an undue state of nervousness is sometimes to be explained by the unsuspected existence of toxic thyroid disease. A patient with such a concurrent disease can readily be reduced to a state of extreme jeopardy and will require the most careful treatment to carry him through what might eventuate in fatal crisis.

#### ADDENDA

Since this chapter was prepared in December 1947, the literature of chemotherapeutic and antibiotic therapy has continued to expand and the following brief notes are presented to bring it up to date.

One death has been reported following penicillin therapy in a 72 year old woman submitted to appendectomy for a gangrenous perforated appendix [Rubinovitch Jacob and Snitkoff Morris C. Acute exfoliative dermatitis and death following penicillin therapy. *J A M A* 138:496-8 (16 Oct.) 1948]. Death which was caused by acute exfoliative dermatitis occurred 7 days after the last injection had been given. Treatment consisted of 400,000 units administered over an 11 day period. It was later found that the patient as well as two of her children was allergic to penicillin. The authors point out that the haphazard use of penicillin is not devoid of risk and suggest that great caution should be exercised in its routine use especially in elderly patients and in patients with a history of allergy.

Recent reports add further testimony to the possible risks of streptomycin therapy. Behrm and Perr reported the occurrence of stomatitis

## ( VIII )

### *Special Types of Acute Appendicitis*

#### I ACUTE APPENDICITIS IN INFANCY AND CHILDHOOD

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##### GENERAL CONSIDERATIONS

Physicians have long recognized that the incidence and mortality of acute appendicitis differ according to age groups. Although proportions differ in different series the 6441 cases analyzed from the records of Charity Hospital of Louisiana at New Orleans over the 15½ year period ending 31 December 1945 are typical of the disproportionate distribution observed in practically all series investigated from the standpoint of age (fig 69). In this series the extremes of life represented well under a third of the total cases but well over a half of the total deaths. Children up to 12 years of age represented 18.6 per cent of the incidence but more than 24 per cent of the deaths. There were also 39 children among the 158 patients who died of acute appendicitis in Charity Hospital without operation over the same period of time.

Comparative studies from the literature are particularly unsatisfactory in children. Some recorded series include only surgical cases. Other series include cases treated by both surgical and nonsurgical measures. Some reports are from children's hospitals, the concentration of cases is therefore not representative and the statistics are frequently also weighted by the fact that treatment is by physicians and surgeons with long experience in children's diseases and is therefore better than average. A prolific source of confusion is the fact that not all series are analyzed on the same age basis. In some childhood is considered to extend through the sixteenth year, which again weighs the statistics for the disease as most frequent in adolescents and young adults and the mortality is lowest in these age groups.

On the basis of the somewhat overworked analogy between the appendix and the tonsil it would seem that the amount of lymphoid tissue in the appendix which increases as childhood advances would make for a greatly increased frequency of appendicitis in children. The incidence of the disease does increase as the child grows older but the increase is disproportionately small compared to the increase in the

higher than are to be expected from single sulfonamides. The combined sulfonamide preparation is now available commercially in tablet and fluid form. If the drugs are administered in combination in dosages of 8 gm per day and the volume of urine is above 1500 cc per day it is not usually necessary to administer an alkali though if these conditions are not met or if there is any doubt as to the renal status it is better to alkalinize the urine to a pH of 7.1 or more.

Aurcomycin an antibiotic derived from a strain of *Streptomyces aureofaciens* has been shown to possess antibacterial activity against numerous gram positive and gram negative bacteria including several species present in appendiceal peritonitis though the drug has apparently not been used for this purpose [Byer Morton S. Schoenbach Emanuel H. Chindler Caroline A. Bliss Eleanor A. and Long Fern H. Aurcomycin Experimental and clinical investigations *J A M A* 135:117-19 (11 Sept.) 1948].

<sup>1</sup> Amigen. Council on Pharmacy and Chemistry. New and Nonofficial Remedies *J A M A* 128:363 (2 June) 1945.

Streptomycin in the treatment of infections. A report of one thousand cases. The Committee on Therapeutics and Other Agents. National Research Council. Keefe, Chester S. et al. *J A M A* 132:411 (7 September) 1946.

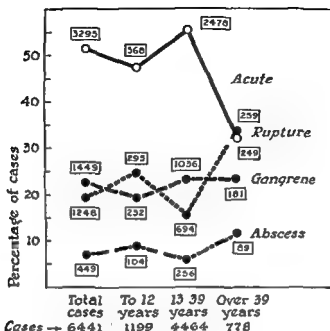


Fig 70 Proportionate distribution of pathologic processes in relation to age in 6441 surgical cases of acute appendicitis

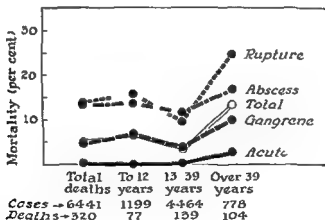


Fig 71 Mortality in relation to age and pathologic process in 6441 surgical cases of acute appendicitis

survived a Ramstedt operation for pyloric stenosis complicated by wound disruption and 5 months later survived an appendectomy for a gangrenous appendix with purulent peritonitis. The high mortality of acute appendicitis in childhood seems much more likely to be due to



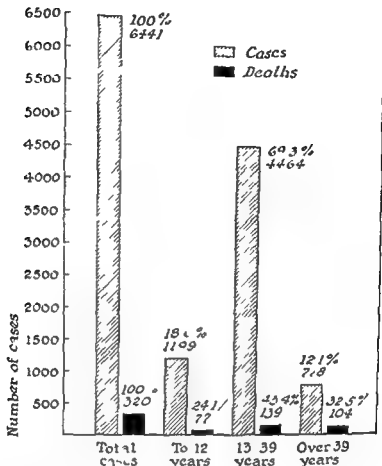


Fig. 69 Proportionate distribution of cases and deaths in relation to age in 441 surgical cases of acute appendicitis

lymphoid tissue and also in comparison to the increased incidence of tonsillitis as childhood advances.

Aside from the anatomic reasons to be outlined shortly, it is difficult to explain why the pathologic process should be so serious in childhood (figs 70-71). Lowered resistance does not seem to be a reasonable explanation. Children actually seem to have a high resistance. Nurslings rarely contract contagious diseases and the Schick and Dick tests show that resistance to diphtheria and scarlet fever is highest in very early childhood. Moreover, surgeons with a large experience in the diseases of childhood are frequently impressed with the remarkable ability of young children to withstand surgical and accidental trauma, as is illustrated by the case reported by Vickers and Conrad. A premature child at the age of 1 month when its weight was only 3 pounds 12 ounces

hood and the more ready interference with the blood supply of the appendix which permits gangrene and perforation to occur rapidly particularly when there is obstruction of the lumen. Furthermore the appendix is relatively long and the cecum is likely to be more mobile than in the adult. Finally, the omentum in childhood is thin delicate short and devoid of fat and therefore offers less protection than it does in older persons. The lack of fixation of the cecum, the relatively large size of the appendix and the relative ineffectiveness of the omentum mean that there is correspondingly less chance of the isolation of an inflamed appendix and of the walling off of infection when the process reaches the serosal coat or when rupture occurs.

Wakley, who analyzed the pathologic process and the prognosis of acute appendicitis in childhood in relation to the position of the appendix, concluded that the retrocecal position is not unfavorable if the disease is limited to the appendix but definitely unfavorable if perforation has occurred and the cellular tissues of the abdominal wall have become involved. If the appendix is long and retrocolic it is difficult to prevent absorption of septic material upward spread and pulmonary complications. If it lies in front of or behind the terminal ileum intestinal obstruction may occur. If it lies in the pelvis the prognosis is fairly good even after rupture. The anterior or splenic position which is fortunately unusual carries a poor prognosis.

*Duration of Illness.* The time of consultation and of hospitalization largely determine both the stage of the pathologic process and the outcome of the illness. Allen's studies show this clearly. In the fatal cases in his series the average time from onset to operation was 102 hours against 53 hours in the nonfatal cases. In the group of cases without rupture the average time from onset to operation was 39 hours against 152 hours for the group with abscess. The most encouraging feature of his special study was that in the second half of the period investigated the average duration of illness before medical consultation was reduced to 47 hours as compared with 59 hours in the first half.

In the Charity Hospital cases in which the duration of illness was stated the mortality increased progressively as the duration of illness before hospitalization increased (fig. 72). In a group of 250 consecutive cases analyzed from the standpoint of pathologic change in relation to duration of illness it was found that at the end of 24 hours over half of the children presented gangrene or rupture with a high percentage of spreading peritonitis. At the end of 48 hours almost 75 per cent presented the same advanced changes. This series like other recorded series suggests that children with acute appendicitis are likely to exhibit

massive infection from early perforation of the appendix, which overcomes normally high resistance thru to my supposed lowered resistance at this age

The point is sometimes made that acute appendicitis is actually no more serious in young children than in older individuals but merely carries a higher death rate because diagnosis is more difficult and complications are more frequent. That point of view seems a distinction without a difference though undoubtedly part of the mortality of acute appendicitis in childhood can be traced to the difficulty of eliciting a complete history. This difficulty in turn is due to the child's inability to describe his symptoms and to the necessity of depending upon physical signs which other than tenderness are notoriously inconstant.

Another consideration that several writers have emphasized is that delay in medical attention which is so striking in many reported series of acute appendicitis in children is regulated by social and economic factors and is partly attributable to the inexperience of young mothers. It is questionable whether much emphasis should be placed upon this factor. Ignorance in such matters is by no means confined to the lower social and economic strata while even the most intelligent of mothers and nurses might readily fail to realize for a period of hours that a very young child's restlessness and crying spells and unwillingness to nurse are related to abdominal discomfort. Whatever the causes the end result is the same that too many young children continue to die of acute appendicitis and that a concerted effort of pediatricians, general practitioners and surgeons is needed to improve the situation.

Regardless of the reasons the proportion of uncomplicated cases of acute appendicitis is small and the proportion of complicated cases is high in practically all reported series of acute appendicitis in childhood. In the Charity Hospital series among 1199 children up to 12 years of age only 568 not quite half had the simple acute variety of the disease (fig 70 p 337). It is significant that only 2 of the 77 deaths in this age group occurred in the acute cases (fig 69 p 336).

### PATHOLOGIC PROCESS

*Anatomic Considerations* Certain anatomic factors seem to play a part in the type of appendicitis observed in young children. At this age because of the funnel shape of the ceco-appendiceal junction the likelihood of free intracecal drainage of material from the appendix is probably greater than at any other age. This advantage however is perhaps offset by the greater delicacy of the appendiceal wall in child

out The rapid sequence of events in the progression of the pathologic process is the reason why in children even more than in adults the mere taking of a purgative should be regarded as an indication for immediate operation and why the cessation of pain after it has been taken which so often indicates the onset of gangrene should be regarded as Murphy expressed it as the last call to operate

### PREDISPOSING AND ETIOLOGIC FACTORS

*Sex and Race* Sex probably plays no part in the incidence of acute appendicitis in childhood The incidence in males is higher than in females in most reported series and the mortality in females is usually though not invariably higher In the Charity Hospital series almost two thirds of the total cases in children occurred in males and the mortality in this group was 5.87 per cent as compared with the mortality of 7.33 per cent in females

The mortality of acute appendicitis in children at the New Orleans Charity Hospital (fig 71 p 337) is unduly high in comparison with other reported series This can be partially explained by the large Negro population at this institution Negro children who often receive very casual care at home partly because of ignorance partly because so many mothers are working women accounted for only 28 per cent of the incidence in children in this series but for more than 35 per cent of the mortality The Negro death rate was 7.96 per cent as compared with the white rate of 5.81 per cent Not many comparative series are available for study but it is significant that in Stone's series of 258 cases in which the mortality was unusually high (7.7 per cent) there were 39 Negroes In Allen's series the death rate in Negroes with acute appendicitis was 17.6 per cent as compared with 5.8 per cent for the whole series

*Age* Acute appendicitis is relatively infrequent but extremely serious in very young children Abt in 1917 collected from the literature 80 cases in children under 2 years of age including 2 cases of possible prenatal appendicitis Additional cases of prenatal appendicitis have been reported by Hill and Mason and by Corcoran Abt's statistics are not entirely clear but it is possible to determine that his series includes at least 11 instances of gangrene 23 of appendicitis with perforation and 10 of appendicitis with abscess formation At least 36 of the 80 children died When the child is premature as in the case reported by Etherington Wilson<sup>1</sup> the hazard is greatly increased

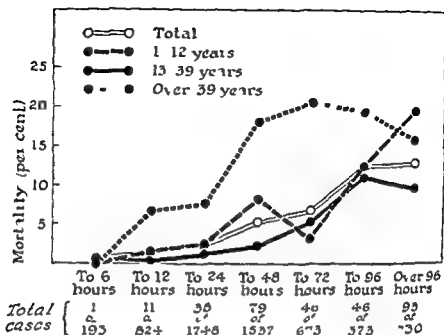


Fig. 72 Mortality in relation to age and duration of illness (symptoms) before hospitalization in 6441 surgical cases of acute appendicitis

more serious and more widespread changes than adults over similar periods of time and that in this age group when the disease has once gained a foothold it tends to develop with terrible rapidity.

**Purgation.** All available statistics show the evil consequences of purgation in children with acute appendicitis. It is known that purgatives had been taken in 383 of the 1199 cases in children up to 12 years of age in the Charity Hospital series. 36 cases (94 per cent) in this group ended fatally. Purgatives had also been taken in 22 of the 36 fatal nonsurgical cases. They had been repeated in 84 of the surgical cases, 18 of which were fatal and in 16 of the fatal nonsurgical cases. In 20 cases in the combined groups they had been given by physicians. Actually purgatives were probably taken in many more cases in the series than these figures indicate and had simply not been recorded.

These proportions are duplicated and exceeded in most reported series of cases of acute appendicitis in children who because of what Moynihan called the phylloanthropic propensities of their mothers and nurses suffer even more from this type of treatment than do patients in other age groups. The administration of a purgative to a young child with acute appendicitis has more serious consequences than at any other period of life because of the anatomic peculiarities already pointed

the 1 199 cases (21.43 per cent) in children at the New Orleans Charity Hospital but the mortality in this group (fig 73) was only 2.33 per cent as compared with a mortality of 6.42 per cent for the whole series and of 7.54 per cent in the 942 children without such a history.

Cumulative studies from this hospital as noted elsewhere (p 412) all show that the death rate is smaller in patients who have had previous attacks of acute appendicitis and others have made the same observation. It may be as has been suggested that parents are on the alert because of previous attacks and seek medical attention earlier but it is questionable whether that explanation would hold for many of the patients of the social strata treated at the New Orleans Charity Hospital. Whatever the reason the statistics in children seem to suggest a relative degree of mildness after the first attack as opposed to the general virulence of acute appendicitis in this age group.

*Previous Illness.* The possibility of a relationship between acute appendicitis and upper respiratory infections is more striking in children than in any other age group as most reported series indicate. One hundred forty nine of the 1 199 children in the Charity Hospital series 11 of whom died developed their disease in the course of an upper respiratory infection. Two others had bronchitis and 5 had pneumonia when they became ill but there were no deaths in these groups.

Brennemann who devoted a great deal of attention to a certain type of abdominal pain nonappendicular in origin associated with throat infections later continued his study from the reverse point of view feeling a natural degree of responsibility for his teaching since it clearly introduced the risk that some cases of acute appendicitis might be incorrectly diagnosed. In a series of (admittedly inadequate) records of acute appendicitis he found 17 per cent of associated throat infection while of 11 consecutive private patients with throat infections 2 presented mildly acute appendicitis and 4 gangrene of the appendix.

It may be as has been suggested elsewhere (p 53) that the same infection localizes at two separate points simultaneously but the explanation does not seem particularly sound. An explanation of the association however is nothing like as important as is the realization that the coincidence is possible. A child with an upper respiratory infection who also complains of abdominal pain should be very carefully observed preferably in a hospital and exploration should be carried out if acute appendicitis cannot be positively excluded. The unnecessary laparotomies performed under these circumstances are less to be regretted than are the disasters which sometimes ensue when exploration is not carried out.

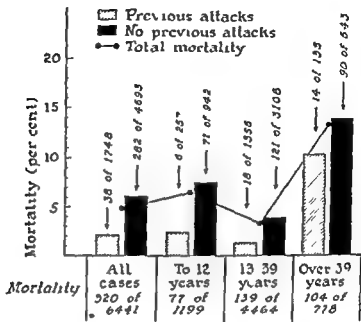


Fig 73 Mortality in relation to previous attacks in 6441 surgical cases of acute appendicitis

This boy who weighed 3½ pounds at birth became ill on the fourteenth day of life and had lost a great deal of weight and was extremely dehydrated when he was seen 48 hours later. After a brief delay for intensive pre operative preparation drainage was instituted. The appendix was retrocecal and gun grinous diffuse peritonitis was present and because the incision was too high the cecum could not be mobilized and appendectomy was technically impossible. Death occurred 48 hours later.

On the other hand appendicitis in infants is not necessarily fatal. The case of Vickers and Conrad (p 336) is an instance in point and Howes and Syphax reported the successful removal of a gangrenous appendix in a 6 day old child born prematurely at 7½ months.

At Charity Hospital 7 of the 11 children under 2 years of age died of acute appendicitis as did 7 of the 23 under 3 years of age and 8 of the 38 under 4 years of age.

**Previous Attacks** Hudson and Chamberlain were inclined to dismiss the question of previous attacks of acute appendicitis in children on the ground that the history is frequently based only on presumption. The comment is no truer about children it would seem than about individuals in other age groups and a history of previous attacks whether presumptive or positive often simplifies the diagnosis of acute appendicitis. A history of previous attacks was secured in only 257 of

the 1 199 cases (21.43 per cent) in children at the New Orleans Charity Hospital but the mortality in this group (fig. 73) was only 2.33 per cent as compared with a mortality of 6.42 per cent for the whole series and of 7.54 per cent in the 942 children without such a history.

Cumulative studies from this hospital as noted elsewhere (p. 412) all show that the death rate is smaller in patients who have had previous attacks of acute appendicitis and others have made the same observation. It may be as has been suggested that parents are on the alert because of previous attacks and seek medical attention earlier but it is questionable whether that explanation would hold for many of the patients of the social strata treated at the New Orleans Charity Hospital. Whatever the reason the statistics in children seem to suggest a relative degree of mildness after the first attack as opposed to the general virulence of acute appendicitis in this age group.

*Previous Illness.* The possibility of a relationship between acute appendicitis and upper respiratory infections is more striking in children than in any other age group as most reported series indicate. One hundred forty nine of the 1 199 children in the Charity Hospital series 11 of whom died developed their disease in the course of an upper respiratory infection. Two others had bronchitis and 5 had pneumonia when they became ill but there were no deaths in these groups.

Brennemann who devoted a great deal of attention to a certain type of abdominal pain non-appendicular in origin associated with throat infections later continued his study from the reverse point of view feeling a natural degree of responsibility for his teaching since it clearly introduced the risk that some cases of acute appendicitis might be incorrectly diagnosed. In a series of (admittedly inadequate) records of acute appendicitis he found 17 per cent of associated throat infection while of 6 consecutive private patients with throat infections 2 presented mildly acute appendicitis and 4 gangrene of the appendix.

It may be as has been suggested elsewhere (p. 58) that the same infection localizes at two separate points simultaneously but the explanation does not seem particularly sound. An explanation of the association however is nothing like as important as the realization that the coincidence is possible. A child with an upper respiratory infection who also complains of abdominal pain should be very carefully observed preferably in a hospital and exploration should be carried out if acute appendicitis cannot be positively excluded. The unnecessary laparotomies performed under these circumstances are less to be regretted than are the disasters which sometimes ensue when exploration is not carried out.



The coincidence of acute appendicitis and other diseases has been discussed in detail in the section on symptomatology and diagnosis (p 157) and all that is necessary here is a reiteration of the statement that the existence of one disease is no protection whatsoever against the possible development of another disease. In the Charity Hospital series coincident disease in children included measles, rheumatic fever, malaria, asthma, whooping cough, mastoiditis, and tuberculosis. Others have reported acute appendicitis coincident with chickenpox, diabetes, hemophilia, mumps, bacillary dysentery, and rheumatoid arthritis.

*Dietary Factors.* Appendicitis that is initiated by dietary indiscretions is particularly confusing and correspondingly serious in children. The figures from Charity Hospital support this point of view. In the surgical series there were 4 deaths in the 23 cases initiated by dietary indiscretions, and 4 of the 39 fatal nonsurgical cases in children were similarly initiated. Whatever the influence of these circumstances may be in respect to the cause of acute appendicitis, there is no question as to their influence on mortality. The potentialities indeed are so grave that if the diagnosis of acute appendicitis cannot be excluded by a reasonable period of observation, the abdomen should be opened without further delay. It seems scarcely necessary to add that in children with gastrointestinal upsets, purgatives should never be given regardless of the apparent indication until acute appendicitis has been duly considered and definitely excluded.

*Parasites.* Although parasitic infection is associated with acute appendicitis in children in a larger proportion of cases than at other age periods, the cause and effect relationship is probably slight (p 53). The possibilities of delay and confusion are however, grave. Infestation was recorded in only 37 cases in children at the New Orleans Charity Hospital, but 5 were fatal chiefly because of delay in diagnosis and the use of vermifuges. Parasites were also present and had the same disastrous consequences in 3 of the fatal nonsurgical cases. Scott and Ware, who reported 4 instances of perforation of the appendix in 25 cases of parasitism associated with acute appendicitis, suggested that in an occasional case a bolus of pinworms might be responsible for obstruction of the lumen.

*Fecoliths.* Acute appendicitis associated with obstruction is as frequent and as potentially serious a type in childhood as in other periods of life. Indeed it is probably more serious because for the reasons already mentioned rupture occurs more readily in childhood. The part played by fecoliths is however difficult to determine because in most series, as in the Charity Hospital series, the figures are incomplete.

Hudson and Chamberlain whose statistics are unusually complete found fecaliths in 43.6 per cent of their cases the distribution being 21 per cent in acute appendicitis without fluid 43 per cent in acute appendicitis with fluid 53.6 per cent in instances of rupture and localized peritonitis 65.2 per cent in instances of rupture and spreading peritonitis and 26.7 per cent in appendiceal abscesses. Rupture had occurred in 58 per cent of all cases in which fecaliths were present. Scott and Ware who continued Hudson and Chamberlain's study (from the Children's Hospital Boston) found fecaliths in 40 per cent of all cases and in 66 per cent of the cases with perforation. The figures leave little doubt of the adverse effects of these concretions.

### CLINICAL PICTURE

*Symptoms* The classical symptoms and signs of acute appendicitis occur in children just as in other age groups but the proportion of atypical cases is considerably higher. Whether this is due to the child's inability to describe his complaints or to really atypical symptoms does not in any way simplify the diagnostic problem.

Gastrointestinal disturbances are generally more frequent in childhood than in adult life and the differential diagnosis is particularly difficult in children habitually subject to such upsets. A child in the Charity Hospital series for instance had had intermittent attacks of diarrhea for several months before an attack of acute appendicitis which was ushered in by the same type of onset and which was recognized as appendicitis only at autopsy. Twenty-eight cases in the surgical series 8 of which were fatal were associated with diarrhea.

Urinary symptoms are perhaps more frequent in children than in other age groups because of the position of the bladder at this time. Pyelitis is also fairly frequent in young children and the physician who must decide whether the current illness is a recurrence of a previous pyelitis or is an attack of acute appendicitis faces a difficult problem. All the unexpected and bizarre symptoms apparently unrelated to appendicitis which are encountered in other age groups are also met in children.

*Vital Signs* The reaction to any disease process is likely to be more rapid in childhood than at any other period of life and the constitutional manifestations may be so marked as to overshadow local symptoms and signs. The temperature and pulse rate both tend to be proportionately higher in this age group though any level may be observed. In

the Charity Hospital series the temperature ranged from  $97^{\circ}$  to  $106.2^{\circ}$  F and the pulse rate from 60 to 180

The respiratory rate in children is normally higher than in adults and while an unusually high rate is suggestive of respiratory disease it is not inconsistent with acute appendicitis. In short in children as in adults the important consideration is that acute appendicitis may exist with any possible variation of the vital signs or with no alteration at all.

*Physical Findings* In the early stages of acute appendicitis the patient looks much like any other sick child. He is frequently restless and fretful and may be screaming with pain. If treatment is not instituted promptly however he soon begins to look very ill. Dehydration occurs early in children deprived of food and fluids who are also losing fluids by vomiting and perhaps by diarrhea. As peritonitis develops the expression becomes anxious and apprehensive and the typical hippocratic facies eventually appears.

Tenderness is the most dependable finding in young children suspected of having acute appendicitis. It is not as simple to elicit as in older persons and it must be interpreted with caution for the child in fear of being hurt is likely to complain when scarcely touched. The usual methods of examination (p. 138) must therefore be carried out with special care. Every endeavor should be made to win the child's confidence before palpation is begun but Christopher and Jennings sensible warning should be remembered that one is not likely to establish a rapport with even a young child by too boisterous a degree of Pollyannaism. It is a distinct advantage though this is not often possible to begin the examination while the child is asleep.

In searching for the point of maximal tenderness in children it should be remembered that McBurney's point at this age lies in the middle third of a line drawn from the anterior superior spine of the ilium to the umbilicus and that the disproportionately deep location of the appendix may mean that tenderness or muscle spasm may be more marked on the left than on the right side and may be considerably higher than usual. Rigidity is frequently absent in children before a peritoneal reaction has occurred especially if the appendix is retrocecal or pelvic.

Since the examining finger can reach higher and therefore feel more in the smaller pelvis rectal examination is often more valuable in children than in adults. It must be carried out with particular gentleness preferably after the child's cooperation has been gained. The complaint of tenderness is perhaps open to question but the finding of a mass is conclusive.

*Laboratory Data* No greater diagnostic reliance can be placed upon the white blood cell count in children than in any other age group. The Charity Hospital series shows a typically wide range from 4 000 to 38 000 per cu mm. The polymorphonuclear leukocyte percentage ranged from 44 to 98. Extremely high counts were most usual in patients with serious complications though this was not universally true. Leukopenia was often of serious import.

Jacobson in a study of 918 children up to 12 years of age was able to trace a definite diagnostic and prognostic correlation between the incidence of peritonitis and the degree of leukocytosis, the associated proportional rise of polymorphonuclear leukocytes and the temperature level. He found no relationship between the leukocytosis and the incidence of appendiceal abscess. The leukocyte pulse rate index aided in determining the degree of resistance of patients with peritonitis. Most observers would hesitate to place great reliance upon the correlation in view of the notorious variability of the white blood cell count in all phases of acute appendicitis.

The possible urinary findings in acute appendicitis have been discussed in detail elsewhere (p 118). Since children do not tolerate starvation well, blood chemical studies should be carried out in all complicated cases as a guide to pre-operative therapy; they are not useful in diagnosis.

### DIAGNOSTIC CONSIDERATIONS

The chief considerations in the diagnosis of acute appendicitis in children have already been stated or implied, but certain general statements may be added. The principal diagnostic difficulty, as already emphasized, is that children cannot describe their symptoms accurately if at all. The history of the illness must therefore be secured from parents and nurses, and its accuracy is related to their experience and intelligence and to the amount of observation and care which the child receives. In public institutions, completely unreliable stories are often secured, since mothers in this social level are often too busy or too ignorant to give their children very much attention until they become seriously ill.

Many observers have commented on another diagnostic difficulty, that children frequently obscure their symptoms by their course of conduct. The average child will often continue to play until his pain becomes disabling. Repeatedly, one secures the history of a child who felt a little sick, had a little pain, perhaps vomited once, then went out

to play or was sent to school sometimes for an hour or two sometimes for a day or two until he became ill again. One is not likely to suspect serious illness in a child who behaves in such a manner yet just such a story was encountered in more than one of the fatal cases observed at the New Orleans Charity Hospital.

The high proportion of atypical cases in childhood has already been commented on. In a number of cases in the Charity Hospital series pain was not a major complaint. In others the initial pain was in the lumbar region, the scrotum or the right leg or was bilateral. Nausea and vomiting particularly vomiting are likely to be more constantly present in childhood than in other periods of life but either or both were absent in 101 of the 1199 patients in this age group at Charity Hospital.

The chief diagnostic difficulties it must be emphasized occur early in the disease. What is an obvious diagnosis to an inexperienced intern in the hospital may have been a very obscure diagnosis several hours earlier even to an experienced pediatrician or surgeon.

The solution of the diagnostic problem is to bear acute appendicitis in mind and to rule it out even in the most unlikely cases. If the diagnosis cannot be made promptly the patient should be examined at frequent intervals over a period not exceeding 6 hours. Food and fluid should be withheld in the interval, no medication should be given and if at the end of the period of observation an exact diagnosis is still not possible exploration should be carried out.

*Differential Diagnosis.* The pitfall of acute appendicitis in adult life Finney Sr. once said is that something else may be mistaken for it while in children the danger is that acute appendicitis may be mistaken for something else. Pneumonia is the disease most likely to be confused with acute appendicitis in childhood and most of what has been said under the general heading of differential diagnosis (p. 163) is applicable to children. One or two points should be particularly stressed. The respiration and pulse are always faster in children than in adults and the constitutional reaction of temperature is more marked so that the elevations in the vital signs must be correspondingly higher to be appreciated. Holland's point of differentiation is perhaps as good as any if the diagnosis cannot be arrived at by more exact means. The child with pneumonia like the child with any other infectious disease will sleep for more or less long stretches but the child with acute appendicitis will neither sleep himself nor let any one else sleep.

Acute mesenteric lymphadenitis which is common in childhood often cannot be differentiated with certainty from acute appendicitis. An

unnecessary exploration usually does no harm since appendectomy in this disease is apparently beneficial (p 173)

Most of what has been said in the section on diagnosis in general (p 137) is fully applicable to diagnosis in childhood. It should be emphasized again however that appendicitis may co-exist with any other disease and that serious consequences may follow if this possibility is not borne constantly in mind. Sometimes if the symptoms are caused by one of the more uncommon diseases diagnosis can be made only by exploratory laparotomy and the wisest plan in doubtful cases is to resort to that measure without great loss of time because of the speed of the progress of the pathologic process in young children. Analysis of large series of cases will show for every laparotomy with no positive findings another instance in which clinical signs and symptoms of the same character and degree of severity were associated with a pathologic process of such intensity that the outcome would almost certainly have been fatal without surgical intervention. The categorical advice is therefore warranted that the surgeon who is in any doubt about the condition of a child's appendix would do well to waste no time in taking it out.

### THERAPY

When the child is seen early in his illness immediate appendectomy is the only procedure to be considered. Even authorities who routinely advocate expectant treatment for appendicitis with peritonitis and abscess for the most part hesitate to employ that type of treatment in children who are not seen until the disease has advanced. Bower stated that he had not found it necessary to treat children differently from adults since they too develop antitoxin in the blood but this is not the usual point of view. The chances are that a child treated conservatively will be overwhelmed by infection and toxemia before satisfactory localization of the pathologic process has occurred.

A. J. Ochsner did not consider delayed operation wise in children because the omentum is not sufficiently developed to be protective. Collier and Potter reported that a high proportion of the deaths in their series occurred in children treated conservatively and raised the question as to the wisdom of this method at this age. Bruce specified that expectant treatment should be practiced in childhood only when there is clear evidence of localization that is when a mass is palpable along the parietal abdominal wall or in Douglas pouch and when *most important* the rest of the abdomen is free from any sign of peritoneal involvement and is soft and lax. The opinion in short is fairly general

that whatever be the advantages of conservative therapy in acute appendicitis with complications at other periods of life it is dangerous in children because of their intolerance to toxemia and their liability to acidosis from starvation. It seems significant that conservative treatment was deliberately elected in 17 of the 30 fatal nonsurgical cases in children at the New Orleans Charity Hospital in which the diagnosis of acute appendicitis was made *intemortem*.

The experience reported by Schulz from the Milwaukee Childrens Hospital is in sharp contrast to the experience of other observers with the nonsurgical treatment of the perforated appendix in children. Over the period 1929-46 inclusive the mortality progressively decreased from 25 per cent in the first 5 years to zero in the last 3 years although during the first 5 years 90 per cent of the children were subjected to immediate operation against only 33 per cent in the last 3 years. The author attributes the increasingly good results to the use of intestinal decompression, maintenance of the fluid and electrolyte balance, multiple small transfusions, chemotherapy and antibiotic therapy and the nonoperative treatment of perforation of the appendix. The duration of illness prior to treatment was substantially the same throughout the period studied. The highest mortality occurred in children admitted to the hospital from 10 to 21 days after their initial symptoms following secondary rupture of an appendiceal abscess into the free peritoneal cavity.

Schulzs results would be remarkably good by any method of treatment but his experience with nonsurgical measures it should be emphasized again is not the usual experience in appendicitis with perforation in young children.

If operation can be carried out swiftly without undue prolongation of the operating time without soiling of uncontaminated areas of peritoneum and with a minimum of trauma appendectomy is as wise in complicated as in uncomplicated acute appendicitis. Otherwise simple incision and drainage would seem the wiser plan though this is never the procedure of choice. Many children in advanced stages of acute appendicitis are exceedingly ill and are in no condition to withstand extra minutes of anesthesia or unnecessary manipulations. Fortunately appendectomy even in an advanced stage of acute appendicitis is frequently a simple matter because of the absence of limiting adhesions.

Ethylene cyclopropane or light ether anesthesia is equally satisfactory. If the McBurney approach is selected the incision should be somewhat higher than in adults and should lie entirely above the line

between the umbilicus and the anterior superior spine of the ileum. One reason is that the appendix is often higher in children than in adults because rotation of the colon and descent of the cecum are often incomplete. Another is that provision must be made for the removal of a possible retrocecal appendix with the tip fixed at the level of the hepatic flexure which may be difficult if not impossible through a low incision.

#### PRE OPERATIVE AND POSTOPERATIVE CARE

Pre operative measures in children with advanced acute appendicitis are similar to those employed in other age groups (p. 293) though a few special precautions are necessary. These patients are frequently approaching a state of acidosis if they have not actually entered it when they are first seen and they are likely to be extremely dehydrated. Sufficient time must therefore be taken before operation to restore the fluid and electrolytic balance by infusion or by transfusions of blood or plasma; the latter plan is recommended by Elman in amounts of 10 to 20 cc per kilo of body weight. Fluid should be administered strictly on the basis of calculated needs for children are easily overwhelmed by an excess. If peritonitis has developed constant intestinal decompression should also be instituted though the Miller Abbott tube because the size of the tube is out of proportion to the size of the child's nostril is not as useful in younger children as in older patients. Oxygen inhalations are frequently of value.

Replacement therapy is continued after operation on the same scientific basis as before operation. Constant suction and oxygen therapy are also continued. Sedatives are given as necessary with the dosage proportionate to the size and weight of the child. Chemotherapeutic and antibiotic agents are used on the same indications as in older subjects though sulfonamides must be employed with particular precautions against toxic reactions which might readily turn the scale against a very sick child.

Wakeley like many other surgeons has declared that a nurse trained in children's diseases is of more value in the postoperative treatment of acute appendicitis than all the drugs in the pharmacopoeia. Part of the low mortality reported by Gatch and his associates in their series of cases in children is undoubtedly due to their scientific replacement therapy but part of it is implicit in their statement. In every case a nurse on constant duty kept the tube open and the suction always in action. The value of an experienced pediatric nurse cannot be over



estimated but such a desideratum is frequently unobtainable even for private patients while in the average public institution it represents a utopian ideal

<sup>1</sup> Appendicitis in the newborn (*Foreign Letters*) *J A M A* 131 478 (1 June) 1916

## (XIV)

### *Special Types of Acute Appendicitis*

#### II ACUTE APPENDICITIS IN MIDDLE AND LATE LIFE

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##### GENERAL CONSIDERATIONS

IN one of the few discussions in the recent American literature of acute appendicitis in individuals advanced in years Miller listed several reasons why the mortality rate in this age group has not shared the improvement observed in some reported series of acute appendicitis in younger persons. These reasons supplemented by certain opinions of my own are as follows:

1 Elderly persons are prone to seek medical advice late—often too late for their own salvation. They frequently live alone or live with relatives or friends whom they do not wish to trouble (or who do not wish to be troubled) with their complaints. They have fixed notions about what ails them and what should be done about it. They have favorite remedies among which purgatives for abdominal discomfort rank high. They do not like to take advice, particularly from young physicians, and they are frequently opposed to surgery.

2 Because appendicitis is increasingly infrequent as life advances, it may be overlooked as a diagnostic possibility even by an experienced physician.

3 The clinical picture of acute appendicitis in advanced age is even more frequently atypical than it is in other periods of life. Diagnosis is therefore even more difficult than it is at other age periods. The disease in addition must be differentiated from a number of other acute conditions as well as from a number of chronic conditions which are frequent in older persons.

4 Perforation occurs early and the course of events thereafter is likely to be atypical. The spread of infection is rapid because of the essential seriousness of the disease and because general resistance is diminished as age advances. Complications are therefore frequent. Conservative therapy is attended with serious risks.

Acute appendicitis in the aged can justifiably be regarded as a special disease, *l'appendicite du vieillard* or *appendicite chez les malades ages*, as Bernard and Jomain and other French writers have

termed it. In view of this fact and in view of the high mortality rate at this period of life it is surprising to find how little attention is paid to the disease in older persons particularly in the American literature. Only a few articles deal with it exclusively and in most reported series it is treated casually if it is mentioned at all. It is scarcely mentioned more over in most textbooks of surgery from which medical students are supposed to learn the facts about this most common of all surgical diseases. The lack of emphasis upon it in fact is one of the very few adverse comments which can be made against the valuable reports on acute appendicitis in Pennsylvania by the Appendicitis Commission of the State Medical Society.

A cogent reason for emphasis upon appendicitis late in life is the increasing proportion of persons in this age group. Census figures show that the number of persons in this country over 65 years of age has almost doubled since 1900 and that the 45 to 64 year old group has increased more than two thirds. The proportion of older persons in the population furthermore is steadily increasing which means that the number of cases of acute appendicitis in the upper age brackets is actually increasing even though the percentage incidence of the disease may remain unchanged. The problems of diagnosis and management are therefore becoming correspondingly more important.

The very uneven distribution of incidence and mortality of acute appendicitis in the New Orleans Charity Hospital has already been discussed (p. 336-7 figs. 69-71) and it will be recollected that the proportions were particularly evident in the group over 39 years of age. The numbers vary in different series reported in the literature but the same general trends are apparent in them all. In Auds series for instance half the deaths occurred in individuals 40 years of age and older. Cutler reported only 5 instances of acute appendicitis 2 of which were fatal in 204 operations for urgent conditions on aged persons. Lipshutz and Schaffer reported a mortality of 22.5 per cent in 40 patients over 50 years of age against a mortality of 2.5 per cent for their whole series of 1171 cases. In Bondarenko's report of 2888 cases of acute appendicitis at the Second Leningrad Medical Institute between 1919 and 1934 only 14.2 per cent of the patients were over 40 years of age. In 1251 cases of appendiceal abscess and peritonitis reported by Totten 21.5 per cent of the incidence but 39.9 per cent of the mortality occurred in patients over this age. These proportions which can be duplicated in almost any reported series suggest that acute appendicitis in middle and late life is deserving of far more attention than it ordinarily receives.

## PATHOLOGIC PROCESS

Every writer who has paid special attention to the subject is aware that there are two reasons for the mortality from acute appendicitis in middle and late life. The first is that the pathologic process *per se* assumes a far more serious character in older than in younger individuals. The second is that the inherently serious character of the pathologic process is enhanced by the character of the symptomatology which is such as to cause delay on the part of the patient and confusion and indecision on the part of the physician.

In the young person acute appendicitis whether it is infectious or obstructive in origin is essentially a suppurative process with perforation and abscess formation the most usual sequelae of untreated disease. The inflammation is at first focal and the whole organ is involved only in advanced stages. In the older subject on the other hand appendicitis is often fundamentally a vascular disease and the initial change tends to be a massive gangrene. This is what might be expected. Vascular degeneration and circulatory impairment are part of the alterations that occur in the mere business of growing older. Arteriosclerosis may play a part in some changes which possibly are of embolic origin. In the older subject the amount of lymph tissue in the appendix is reduced and infection as a primary process plays a less important role. Finally the circulation of the appendix furnishes an appropriate background for this sort of pathologic change.

In the young subject particularly in the adolescent and the young adult acute appendicitis often shows a tendency to localize. In the older subject it has a tendency to spread. Localization while it may occur after rupture is by no means the rule and a diffuse or spreading peritonitis is correspondingly frequent.

The vascular character of acute appendicitis in subjects of advanced years is often evident at operation. Tortuous veins are visible in the meso-appendix and mesentery. The adjacent structures are likely to be inflamed and injected. The appendix itself exhibits large areas of gangrene or even massive gangrene rather than the small patches or microscopic areas more frequent in younger persons. Sometimes the appendix has been amputated and lies free in the cavity or it may fall to pieces in the process of removal. The gangrene may have spread to the meso-appendix or even the cecum. The tissues may be so friable that sutures break through them and the control of hemorrhage is a real problem. Retrograde thrombosis is sometimes observed. It is not surprising in

view of these facts that aged subjects exhibit far oftener than young subjects such complications as mesenteric thrombosis, pyloritis, or the metastatic liver abscess first described by Dickroff as the appendicular liver.

These findings naturally are not uniform. Many cases of appendicitis in the aged do not differ in respect to the pathologic process from acute appendicitis earlier in life. The type of change described however is very frequent and is perhaps the chief reason why acute appendicitis late in life is so serious and so often fatal.

An examination of the Charity Hospital series bears out what has been said (fig. 70 p. 337). More than two thirds of the cases of acute appendicitis in individuals over 39 years of age had passed beyond the acute stage when the patients were first seen as compared with 44.5 per cent in the middle group and 52.6 per cent in children under 12 years of age. Even when the patients in the older age group were seen within 24 hours of the onset of illness the pathologic process had frequently advanced to the stage of rupture and gangrene and the mortality for this period (fig. 72 p. 340) was 6.47 per cent as compared with 2.0 per cent in children under 12 years of age and 1.0 per cent in the 13-39 age group. In 100 consecutive patients 40 years of age and over studied from the standpoint of pathologic change in the Charity Hospital series there were only 23 cases of acute or acute suppurative disease though 27 patients had been operated on within 24 hours and 22 others within 48 hours of the onset of illness. Gangrene was the outstanding pathologic change in 60 of the 100 cases; it was associated with rupture in 12 cases and with rupture and spreading peritonitis in 16. Other writers report similar findings. Tamamann and Lohmann for instance studying 73 cases of frank peritonitis in this age group found that 24 patients had been ill only 24 hours and 25 others had been ill only 48 hours.

It is scarcely necessary to add that everything which has been said about the dangers of purgation in acute appendicitis in other age groups can be said with double force of the disease late in life.

### CLINICAL PICTURE

*Symptoms.* Quite as striking as the seriousness and rapid spread of the pathologic process in many cases of acute appendicitis late in life is the character of the symptoms which are indeed frequently responsible for the advanced state of the disease when the patient is first seen. As has been emphasized elsewhere (p. 122) the classical

syndrome is absent in so many cases of acute appendicitis that reliance upon it for diagnostic purposes at any period of life can result only in disaster. It is a particularly grievous error to rely upon it in middle and late life when to adapt Howard Kelly's aphorism about ectopic pregnancy the most typical thing about the clinical syndrome is that it is atypical.

In the young individual an attack of acute appendicitis is often characteristically sudden. A perfectly well individual suddenly becomes an acutely sick individual. In later life on the other hand the patient as Lazarus well expressed it enters the state of ill health slowly. The disease tends to begin in advance of the acute attack sometimes considerably in advance with a period of vague digestive distress which may be associated with diarrhea. The initial pain is frequently little more than discomfort. It is commonly referred to the umbilicus or epigastrium but may be located in the pelvis. The process of localization in the right side may take days in contrast to hours for the similar process in younger persons. Many times it does not occur at all. There were no localizing signs in 41 patients 40 years of age and over operated on at the New Orleans Charity Hospital 15 of whom died or in 12 of the 42 nonsurgical deaths in this age group.

Left sided pain is comparatively frequent in appendicitis in the aged and may be due to the upward displacement of distended coils of ileum. The dangerous period of calm after the appendix has ruptured or become gangrenous may last for intervals varying from 24 hours to a week or more. During this period the patient may be actually comfortable or only mildly uncomfortable. Several patients in the Charity Hospital series left the hospital during it because they regarded themselves as recovered or were discharged by the attending physician or were refused admittance on the ground that there was nothing the matter with them. The patient is fortunate who presents obstructive symptoms and cramping pains which even if they are less severe than they would be in younger persons force him to seek relief without too great delay.

The first symptoms are often entirely atypical and unrelated to the appendix. In the Charity Hospital series they included headache back ache malaise rectal hemorrhage scrotal pain and hiccoughs among others. Overindulgence in alcohol or dietary indiscretions preceded the illness in several instances and seriously confused the diagnosis. One Negro just before the onset of his symptoms had eaten a heavy meal of pork chops turnip greens gin and port wine and then had taken a dose of salts. Another had had a midnight meal consisting of pork

chops a whole coconut pie and a bottle of beer. Both had perforated appendicea when they were operated on within 24 hours of the onset of symptoms and both lost their lives.

Vomiting may be the initial symptom, may occur once or a number of times, or may not appear at all. Nausea is rather more frequent and is likely to be persistent. In the 108 cases at Charity Hospital in which either nausea or vomiting or both symptoms were lacking, there were 15 deaths, 13.9 per cent. Diarrhea, either as an initial or a later symptom, seems of particularly serious import in older subjects. It was present in 31 cases in this series, 4 of which were fatal.

**Vital Signs.** Individuals advancing in years, as has been pointed out (p. 126), react more slowly to pathologic processes than do young persons, and elevations of temperature and pulse rate are therefore less marked. In the Charity Hospital series a wide range was apparent in both. The temperature ranged from 96.5° to 106.2° F. in all cases and from 97° to 106.2° F. in the fatal cases. The pulse range was from 46 to 160 per minute in all cases and from 53 to 160 in the fatal cases. In almost a third of the cases, representing all degrees of involvement, the temperature did not exceed 99° F., and in well over half of all cases the pulse rate did not exceed 90 per minute.

**Physical Findings.** Physical findings in acute appendicitis in the aged are notably scanty. Often the patients do not look ill, or their appearance is not different from their usual appearance. They frequently insist that there is nothing much the matter with them. Old persons, however, do not tolerate toxemia well, and sometimes even when the disease is not of long standing, by the usual criteria, the faces are hippocratic, the tongue dry and furred, and the breath offensive. Dehydration occurs rapidly as the result of vomiting and diarrhea, and because the fluid intake is reduced.

Ptessure pain is probably the most reliable physical finding. A uniform soft distention is quite common. Rigidity is sometimes present in the right side, as in younger subjects, but frequently is absent. Lehmann, who derived his experience from an old people's home, pointed out that this would be expected, the defense mechanism in older persons being unreliable because the musculature is flaccid, particularly in bedridden subjects, while obesity may add to the difficulties of the examination. Obesity incidentally is a definite factor in the mortality. In 18 of 43 fatal cases reported by Holman the patients were obese, and there were 13 deaths in 25 obese patients over 59 years of age in the Charity Hospital series.

**Leukocytosis.** In view of the sluggish response of older persons to

constitutional conditions it would not be expected that the white blood cell count would be particularly helpful. The statement sometimes made that it tends to be around 15 000 seems to have no basis in fact. The range in the Charity Hospital series was from 3 775 to 33 000 per cu mm in all cases but the count was under 15 000 per cu mm in almost two-thirds of all cases and in more than two thirds of the fatal cases in which the blood was examined. Leukopenia seems of particularly serious import in aged subjects. The polymorphonuclear leukocyte percentage was from 41 to 98 in all cases and from 60 to 96 in the fatal cases.

### DIAGNOSIS

The history and clinical findings of acute appendicitis in older persons are frequently suggestive of almost any disease except appendicitis. Brunn stated that pelvic appendicitis in this age group suggests diverticulitis first of all. Often the syndrome is compatible with intestinal obstruction not of the acute but of the chronic variety or of the insidious variety caused by neoplasm. The finding of a mass which later proves to be an encapsulated appendix or an appendiceal abscess frequently lends support to the diagnosis of neoplasm. One patient in the Charity Hospital series whose right sided mass was found at autopsy to be an appendiceal abscess was elaborately prepared for operation for carcinoma of the cecum but death ensued before the abdomen was opened.

In the Charity Hospital series the diagnostic possibilities considered in the cases of appendicitis in middle and late life included tuberculous peritonitis gastro enteritis various types of cardiac and renal disease liver abscess amebiasis malaria cholecystitis pneumonia carcinoma of the cecum colon and stomach carcinomatosis of undetermined origin Meckels diverticulum pelvic inflammatory disease ovarian cyst with twisted pedicle ruptured peptic ulcer hypoglycemia pancreatitis prostatitis renal calculus bacillary dysentery mesenteric thrombosis and strangulated hernia for which 1 patient was actually operated on without discovery of the appendiceal involvement. Incredible as many of these diagnoses may seem when they are brought together in a list there was usually justification for all in the cases in which they were made. The diagnosis of acute appendicitis was missed altogether in some 15 per cent of the cases in the older age group at Charity Hospital as compared with 10 per cent in Tamamann and Lohmann's series and 38 per cent in Wood's series (over 60 years of age) but in most instances operation was performed fairly promptly because the disease was supposed to be some other acute abdominal state.



In a large number of instances involvement of the appendix was recognized as such but was considered to be chronic subacute or subsiding acute rather than acute. Three cases in which a note was made that the patients did not seem acutely ill all ended fatally. All the evidence in short suggests that in middle and late life the clinical picture of acute appendicitis as Bumm expressed it is conditioned by advanced age and by the loss of vitality in the tissues.

History taking is often complicated by the deafness mental impairment or actual senility of patients advanced in years and the physical examination is likely to be conducted without co operation for these reasons or merely because of the obstinacy old persons so often exhibit. All the circumstances thus add up to an extreme degree of diagnostic difficulty.

Diagnosis is additionally difficult in persons in the upper age brackets because of the frequent presence of other conditions such as pulmonary cardiac biliary tract and renal disease and hernias. DeTarnowsky emphasized that it is imprudent to base a diagnosis of abdominal disease in the aged entirely on the previous history however relevant it may seem and cited an illustrative case of supposed coronary thrombosis which proved to be acute appendicitis. Two cases in the nonsurgical group of deaths at Charity Hospital support this warning. One woman in the hospital for many months for decompensated cardiac disease and another hospitalized for many years for pulmonary tuberculosis both died without any suspicion of acute appendicitis being aroused partly because the symptoms were vague and unlike the classical picture partly because all the attention was centered on their chronic ailments. Another patient was believed to have nephritis which he actually had and perhaps a temporary incarceration of the inguinal hernia which he had had for several years and which had previously been incarcerated several times.

Three particularly striking cases of this kind were reported by Stalker. One patient developed acute appendicitis in the hospital after amputation of a leg. Another patient who also had Parkinson's disease and bilateral hernias developed it in the hospital after two cardiovascular accidents. The third patient who was ambulatory was under treatment for prostatitis. He developed symptoms immediately after prostatic massage and delayed 48 hours in the meantime taking a dose of salts before he returned to the hospital. These cases which could be duplicated from the Charity Hospital series go to show again that the mere existence of one pathologic state is no protection whatsoever against the development of another.

Attempts to classify acute appendicitis in the aged into special clinical groups have been made by several observers. Bernard and Jomron in addition to the classical cases described a gangrenous variety, a pseudoneoplastic variety and a pseudo occlusive variety. Frankau<sup>1</sup> at a meeting of the Section of Surgery of the Royal Society of Medicine in 1939 described two special types. In one the symptoms resemble intestinal obstruction. The onset is slow with an acute indigestion pain referred to the epigastrium or less often to the right iliac fossa, persistent pyrexia, indefinite physical signs other than abdominal distention, sometimes visible peristalsis and generalized tenderness greatest in the hypogastric and umbilical regions. At operation which these patients tolerate well there is little peritoneal exudate, the intestines are sticky and red and the appendix is seldom walled off by adhesions. In the second type described by Frankau there is an acute attack of indigestion followed by inability to take food, constipation or diarrhea and persistent slight pyrexia. Examination is at first indefinite but later reveals a hard tender mass in the right iliac fossa which increases in size very slowly and which proves at operation to be an appendiceal abscess.

The wisdom of attempting to classify acute appendicitis in aged persons into any special groups seems somewhat doubtful. By far the safer plan is for the physician to be aware of its multiple manifestations and of its atypical character at this age and to bear it constantly in mind not only when he is confronted with acute abdominal disease but also when he encounters a patient with chronic complaints referable to the abdomen and in particular associated with the digestive processes.

### THERAPY

The general therapeutic considerations that apply in acute appendicitis in other age groups also apply to the disease in the upper age brackets. The treatment is immediate operation, the sooner the better. If the diagnosis cannot be made without undue delay, prompt exploration is even more justified at this period of life than at other periods because the consequences become serious so rapidly. If the appendix proves not to be the source of the illness, the surgeon need not blame himself. In advising operation he has taken the only prudent course. There is never the smallest reason for refusing operation to a patient whom the mere act of operation would not kill merely because he has reached a certain age. A person of any age may be so ill that operation must be deferred until measures have been instituted to improve his condition but age in itself is not a contra indication to operation.

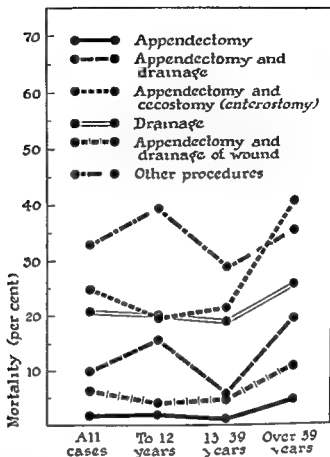


Fig 74 Mortality in relation to age and operative procedure in 6 441 surgical cases of acute appendicitis

If the appendicitis is of the acute variety or if it is at all possible to remove the appendix without undue trauma in complicated cases appendectomy should be carried out. Otherwise the operation should be limited to drainage. Persons of advancing years tolerate prolonged and traumatic manipulations poorly and there is fairly general agreement that if adequate drainage is provided the retention of the appendix alters the prognosis less in this age group than in any other. According to Stalker aged persons seem able to make one effort to localize the infection but are incapable of more if therefore adhesions are disturbed by manipulations the infection will continue to spread even though its source has been removed. The mortality generally speaking is usually dependent not so much upon the special procedure employed as upon the type of lesion that demanded it. Cecostomy and

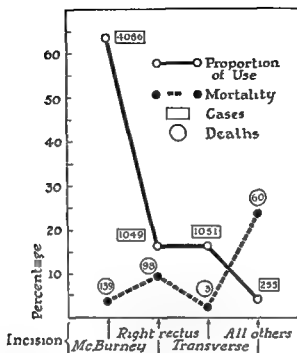


Fig 75 Proportion of use of various incisions with related distribution of mortality in 6441 surgical cases of acute appendicitis

similar operations are no longer used since the introduction of intestinal decompression but it is significant that in the period when this operation was popular in the Charity Hospital cases it was employed proportionately far more often in the upper age group than in any other (fig 74)

The incision depends upon the surgeon's desires. It is worth mentioning that at the New Orleans Charity Hospital where the McBurney incision is the procedure of choice with most surgeons the percentage of other incisions (fig 75) was much higher in the group of patients over 39 years of age than in other age groups. The disproportion is another commentary on the difficulties of diagnosis in later life.

Anesthesia is a matter of no particular consequence if as Johns has expressed it the first misstep of profound anesthesia be avoided. It should be remembered however that spinal analgesia is no safeguard against the respiratory complications to which old people are peculiarly subject after operation and that it may have other undesirable consequences in advanced age.

Conservative therapy should be used with even more circumspection

in individuals advanced in years than in other age groups. In fact if the surgeon is not absolutely certain that localization is definitely progressing immediate operation is by far the wiser plan in all cases for two reasons. Old persons do not tolerate toxemia well and the physician can be even less certain of what is occurring within the abdomen in them than in younger subjects. Another argument against conservative therapy is that it is even more difficult for psychologic reasons to make older persons return for operation than it is to make younger persons. It was often difficult to determine from the Charity Hospital records whether operation was delayed because the surgeon wished to use conservative therapy or merely because he could not make a diagnosis. Whatever the reason for delay the mortality associated with it was high. Collins found that when expectant treatment was used in a group of patients over 40 years of age the mortality was 37.2 per cent higher and the morbidity 74.8 per cent higher than in a similar group treated by immediate operation. Even Love who practiced expectant treatment routinely did not consider it suitable in advanced age.

#### PRE OPERATIVE AND POSTOPERATIVE MANAGEMENT

Operation should always be deferred in individuals advanced in years until dehydration and perversion of the blood chemistry have been corrected by the usual measures. Its postponement for several hours is fraught with no such dangers as is operation on a patient who has been ill for several days, who is toxic and dehydrated and who may be suffering from some chronic disease or even from some serious organic disease as well as from acute appendicitis. Disregard of these precautions is a fruitful source of postoperative complications in this age group.

The postoperative care of individuals advanced in years is similar to that of individuals in other age groups (p. 293) though a few special precautions are necessary. Morphine should be used with caution and withdrawn as soon as possible as in many older persons whose vitality is reduced its effect is undesirable. Furthermore it is not so greatly needed because their reactions are frequently sluggish; pain is often borne better by older than by younger subjects.

Chemotherapy and antibiotics should be used according to the indications and with special precautions to prevent reactions. The mortality, however, does not show the reduction in this age group which is observed when this form of therapy is used in younger persons. The prevalence of gangrene in this age group may furnish part

of the explanation for such therapy is least effective when tissue necrosis is part of the pathologic process

*Replacement Therapy* The maintenance of fluid equilibrium in the older patient is extremely important. Patients in this age group tend to respond to trauma and hemorrhage with a shock like reaction and do not withstand well periods of hypotension regardless of the cause. Fluid losses must therefore be replaced promptly. On the other hand patients in this age group are likely to have a decreased cardiac reserve and cannot cope with amounts of fluid that increase the blood volume much above the usual level. Many observers believe that it is a wise plan to use infusions (or transfusion if blood loss is more than minimal) during all major surgical procedures in elderly persons so that blood and plasma losses are compensated for immediately.

After operation if the fluid and electrolyte balance can be maintained by mouth the oral route should be used. If it cannot be the parenteral route should be used the intravenous route for practical reasons being preferable to the subcutaneous except in patients known to have a poor cardiac reserve. Factors of safety in the administration of fluid by infusion depend upon the rate of administration a continuous slow infusion permits the administration of adequate amounts with minimal risk. Koop has recommended a combination of hypodermoclysis and intravenous infusion with half or more of the fluid given subcutaneously.

The warning of Collier and his associates against the administration of excessive amounts of salt should be sedulously heeded. Unless there has been an excessive chloride loss or unless plasma chloride determinations indicate a deficit no salt solution at all should be administered on the day of operation except the small amounts necessary to start a transfusion and on succeeding days administration of physiologic salt solution should not exceed one liter. Colloidal infusions of blood plasma or plasma substitutes should be in small amounts repeated if necessary this plan being safer than the administration of the same total amount in single infusions. Moreover infusions of such substances should be discontinued as soon as the desired response is secured if an excessive quantity is given patients with decreased cardiac reserve are in real danger of developing pulmonary edema. In this connection Koop has made the practical suggestion that colloidal infusions be given during the day rather than at night because more nurses and medical personnel are then on duty and emergencies such as pulmonary edema are likely to be promptly recognized and properly treated.

*Change of Position* It is most important that the position be changed

at frequent regular intervals. The nurse should move the patient after operation until he is able to move himself after which he should be encouraged to take passive exercises. All older patients do better if they are got out of bed or at least are put into a semi-recumbent position as soon as possible because hypostatic pneumonia is a frequent cause of death after operation in this age group. Patients with constitutional disease should be under the care of a competent internist before operation and his services should be continued throughout the postoperative course. Thrombosis and embolism are particularly frequent in this age group (p. 248) and early movement and other precautions are essential to prevent them.

### CAUSES OF DEATH

Convalescence in the upper age group is seldom as smooth as in younger subjects. Appendicitis itself is often more serious. Often it is complicated by organic disease. Aged subjects confined to bed are particularly prone to develop pulmonary or vascular complications. They do not tolerate toxemia well. The proper fluid balance as has been noted is particularly difficult to maintain because there may be a conflict between the patient's needs and the amount of fluid that can safely be administered.

The causes just listed account for many of the fatalities in acute appendicitis in aged subjects but not for all. Many times there is no specific reason for the fatal outcome. The patients do badly simply because they lack the strength or the will to live or perhaps as has been suggested because their day is done and they know it.

Specifically the cause of death in aged subjects is chiefly peritonitis just as it is in all age groups. Vascular and embolic causes play a part in many cases which is not unexpected in view of the dominant vascular factor in appendicitis and the prevalence of vascular disease late in life. The number of such deaths would undoubtedly be increased if post mortem examination could be secured in all cases. Subphrenic abscess is no more frequent in aged patients than in young subjects in whom at the most it is not very frequent but pylephlebitis is relatively more frequent because of the vascular character of the disease. The proposal that ligation of the affected vein be done whenever frank suppuration is seen to exist at operation or seems likely to occur was never widely employed for prophylaxis in advanced age involves a degree of risk against which the possibilities of the development of the complication

must be weighed (p 227) Chemotherapeutic and antibiotic agents have eliminated whatever usefulness the method ever possessed

In some instances the patients survive appendicitis but die because of their background of organic disease Such deaths of course must be counted as ultimately due to appendicitis In any series however the number of deaths due to pneumonia or vascular complications or to cardiac or renal disease will clearly reflect the precautions taken—or not taken—to prevent them

### CASE REPORTS

The following cases are cited as illustrating the atypical clinical picture the difficulties of diagnosis and the advanced pathologic changes found in acute appendicitis in persons beyond middle life

*Case 28* A very obese white woman 45 years of age had had qualitative indigestion for a number of years Three days before hospitalization she experienced epigastric pain accompanied by flatulence and belching and followed 2 hours later by vomiting The pain localized in the right side within a few hours but when she was admitted to the hospital only soreness was felt The temperature was 100.4 F the pulse rate 92 and the respiratory rate 24 The patient presented generalized abdominal tenderness more marked in the right upper and lower quadrants and generalized distention Tenderness was also elicited in the right flank Rectal examination revealed bilateral tenderness The white blood cell count was 15,750 per cu mm with 84 per cent polymorphonuclear leukocytes Urinalysis revealed occasional hyaline casts X ray examination of the abdomen by flat plate showed multiple fluid levels

The admission diagnosis was acute appendicitis versus acute cholecystitis Operation which was carried out at once under ether anesthesia through a right rectus incision revealed a gangrenous appendix and generalized peritonitis Appendectomy cecostomy and drainage were performed The patient died of peritonitis on the fourth postoperative day

The previous history of qualitative indigestion and the posterior localization of the tenderness made a diagnosis of acute cholecystitis seem quite reasonable in this case The subsidence of the pain should be noted It should also be noted that generalized peritonitis developed in the absence of gross rupture as frequently happens in older subjects

*Case 29* An obese white man 56 years of age had suffered from vague digestive symptoms for several days Two days before admission to the hospital he developed a severe cold and 24 hours before admission he began to have epigastric pain The temperature was 99 F the pulse rate 104 and the respiratory rate 20 Physical examination revealed bronchitis a small umbilical



hernia and generalized abdominal tenderness possibly more marked in the right lower quadrant. Rectal examination was negative. The white blood cell count was 18 900 per cu mm with 87 per cent polymorphonuclear leukocytes.

Laparotomy through a right rectus incision under spinal analgesia revealed a ruptured gangrenous appendix with generalized peritonitis. Appendectomy and cecostomy were performed and drainage was instituted. Death occurred on the tenth postoperative day from bronchopneumonia.

This patient had digestive symptoms before the symptoms of acute appendicitis developed. The absence of nausea and vomiting and of localization and the speed of the pathologic process should be noted. The illness developed while the patient was suffering from an upper respiratory infection and the use of spinal analgesia did not prevent death from pneumonia.

*Case 30* A white man 44 years of age suffered from nausea, vomiting and abdominal pain for 4 days following a drinking bout which had lasted for several days. The day of admission to the hospital his acute pain was replaced by epigastric soreness and a sense of breathlessness. Chills had occurred the day before admission. The temperature was 101.8° F, the pulse rate 130 and the respiratory rate 36. The abdomen was extremely rigid and there was slight generalized tenderness. Tenderness in the midline was elicited on rectal examination. The white blood cell count was 11 000 per cu mm with 88 per cent polymorphonuclear leukocytes. Urinalysis revealed a slight trace of albumin.

Appendectomy and drainage were carried out under spinal analgesia through a McBurney incision. The appendix was ruptured and gangrenous and a generalized peritonitis was present with absolutely no tendency toward localization. The patient died of peritonitis and toxemia on the third postoperative day with an axillary temperature of 107° F.

The onset of symptoms following a drinking bout, the complete absence of localization, the chills and the subsidence of pain after rupture should be noted. The low white blood cell count in the presence of such extensive involvement should also be noted.

*Case 31* A Negro woman 65 years of age had suffered for 14 days with anorexia and cramps in the lower abdomen and for 7 days with diarrhea and vomiting after the ingestion of any food. The temperature was 100° F, the respiratory rate 20 and the pulse rate 90. The patient seemed subacutely ill. Physical examination revealed generalized abdominal distention and diffuse lower abdominal tenderness. Pelvic examination revealed a right ovarian cyst. Rectal examination was negative. The initial diagnosis was carcinomatosis versus possible ovarian cyst. Later a medical consultant suggested a diagnosis of hypoglycemia versus ileus versus ruptured appendicitis and ovarian cyst.

The patient died on the third day of hospitalization and postmortem examination revealed a gangrenous appendix appendiceal abscess and generalized peritonitis

There was nothing suggestive of the classical picture of appendicitis in this case. The first symptoms were related to the digestive process and the lack of localization should be noted

*Case 32* A Negro man 50 years of age had been ill for 36 hours with acute abdominal pain nausea and vomiting. The condition was diagnosed as locked bowels by a physician who advised salts. When the first dose was vomited the physician advised repetition of the medicine until it was retained. In all 6 tablespoonfuls of epsom salts were taken. The temperature was 99 F the pulse rate 108 and the respiratory rate 20. The patient looked acutely ill. The abdomen was distended and diffusely tender and an occasional peristaltic wave was observed. Rectal examination revealed bilateral tenderness. The white blood cell count was 23 600 per cu mm with 92 per cent polymorphonuclear leukocytes. Roentgenologic examination of the chest showed the right diaphragm to be elevated. Flat plate of the abdomen revealed multiple fluid levels. Barium enema revealed no filling defect in the lower bowel.

The patient was treated conservatively on a diagnosis of spreading peritonitis following intestinal obstruction and died on the sixth day of hospitalization with a temperature of 106.2 F. Postmortem examination revealed a ruptured appendix and generalized peritonitis.

The treatment of supposed intestinal obstruction by repeated purgation is evidence of the need for education within the profession in the management of acute abdominal conditions. When the patient reached the hospital it was impossible to determine the origin of the peritonitis. The treatment however followed the lines of expectant treatment in peritonitis.

*Case 33* A white man 59 years of age was awakened from sleep 36 hours before admission to the hospital by nausea and vomiting. An hour later he had a chill which lasted for 4 hours. Several hours after the chill pain was experienced in the lower abdomen and he had high fever. Twelve hours before admission he had vomited again. Hiccoughs had been present intermittently since the onset of his illness and he had had several bowel movements. He had had a cough for several months. The temperature was 99 F the pulse rate 88 and the respiratory rate 32. The patient did not seem acutely ill. Physical examination revealed bronchovesicular breath sounds impaired resonance over the right base posteriorly an occasional ectopic heart beat moderate generalized rigidity slight tympany and a large indirect hernia on the left side generalized abdominal tenderness more marked on the right side slight.

The white blood cell count was 26 500 per cu mm when the patient was

admitted to the hospital and 24 hours later was 21 750 The respective polymorphonuclear leukocyte percentages were 90 and 92 Urinalysis showed a trace of albumin and occasional casts X-ray examination of the chest revealed nothing abnormal Blood chemical studies were within normal range

The admission diagnosis was bronchopneumonia with diaphragmatic irritation versus acute appendicitis with localized peritonitis Twenty four hours later the latter diagnosis was decided upon and the abdomen was opened through a right rectus incision under spinal analgesia Appendectomy cecostomy and drainage were carried out The appendix was gangrenous and ruptured All the adjacent tissues were extremely friable The peritoneal cavity contained free pus and there was an especially large collection in the cul de sac Routine measures for peritonitis were carried out after operation but the patient died within 48 hours

The history in this case was entirely atypical as it frequently is in persons in middle age and later in life and the patient did not seem acutely ill The massive gangrene of the appendix and the friability of adjacent structures are typical of appendicitis in the older age group as is the rapidity of the spread of the pathologic process throughout the peritoneal cavity

<sup>1</sup> Acute abdominal conditions (Foreign Letters) *J A M A* 112 2545 (17 June) 1939

## (XV)

### *Special Types of Acute Appendicitis*

#### III APPENDICITIS DURING PREGNANCY

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Kelly and Hurdon in 1905 wrote that the association of appendicitis and pregnancy was still too infrequent to warrant much consideration and English speaking physicians apparently continued in that opinion for many years. At any rate when Marbury reviewed the subject in 1933 he found that two thirds of the articles were in the foreign literature. In the last decade increasing attention has been paid to the association of appendicitis and pregnancy and the recorded cases in the English and American literature now run into the hundreds.

#### INCIDENCE

The reported incidence of acute appendicitis during pregnancy varies widely and it is not always clear whether only the acute disease is included in all the estimates. Baer, Reis and Arens found only 28 cases among 16,543 deliveries and 1,700 (adult) appendectomies at the Michael Reese Hospital which makes the incidence of appendicitis in pregnancy 0.17 per cent and of pregnancy in appendicitis 1.6 per cent. Smith and Bartlett found 16 cases of acute appendicitis among 66,431 deliveries at the Boston Lying In Hospital an incidence of 1:3,321. 4 other cases which they reported were apparently not acute. Cosgrove found only 18 acute cases among 25,000 live births at the Margaret Hague Maternity an incidence of 1:1,380. It is significant that in the same series of deliveries there were only 7 other surgical emergencies of all kinds. Meiling, over a 13 year period at the University Hospitals of Cleveland found 26 instances of acute appendicitis in 49,681 deliveries a ratio of 1:1,910. Over the same period there were 74 other abdominal complications of pregnancy (exclusive of 158 instances of ectopic pregnancy) but most of them it would seem did not represent acute surgical emergencies as did the appendicitis cases.

Only 23 cases in which acute appendicitis developed in pregnant women were found in the 6,441 surgical cases of the former condition.

studied at the New Orleans Charity Hospital over a 15½ year period. Four occurred coincident with or immediately after abortion, 2 during labor and 3 during the puerperium. The remaining cases, 2 of which were fatal, occurred during gestation. There were 2 instances of pregnancy in the 158 fatal nonsurgical cases of acute appendicitis studied at Charity Hospital over the same period, and a third case in this group developed after an abortion which was probably criminal. High incidences are occasionally reported but for the most part as in these series the association of the two conditions is fractional.

This is a rather curious state of affairs. Acute appendicitis is most frequent in the adolescent and early adult years and the majority of pregnancies occur in early adult life. It would seem therefore that instead of the low incidence reported for the combined conditions a high incidence would be likely. Unless pregnancy confers some inexplicable immunity to acute appendicitis on the pregnant woman which is unlikely, there seems no explanation for the infrequent occurrence of the disease at this period.

#### ETIOLOGY AND PATHOLOGY

A study of the recorded cases of acute appendicitis in pregnancy shows that half or more occur in women who have had previous attacks. Some authors in fact state that an exacerbation of chronic symptoms if not an actual acute attack can be expected during pregnancy in most women who have had previous attacks. Why this should be is not clear though several possible explanations, not all of which are entirely convincing, have been advanced. One is that the increased incidence of constipation in pregnancy results in an increased virulence of *Bacillus coli* and an increased formation of fecaliths. Constipation however has never been proved to be a cause of acute appendicitis. Another explanation is that the pregnant woman has a lessened resistance to infectious and toxic processes. The majority of cases of acute appendicitis however are now believed to be obstructive rather than primarily infectious and toxemia plays no part at all in early cases. Parity also seems to play no part, the distribution in the recorded series apparently being entirely accidental.

The most reasonable explanation of the exacerbation of a previous inflammatory process in the appendix and of the serious character of the disease in pregnancy is the altered anatomic relationships introduced by the latter state, chiefly changes in the position of the cecum and compression of the appendix. Baer, Reis and Arens who studied

78 patients by X ray observed that the appendix undergoes progressive displacement upward after the third month and reaches the level of the iliac crest by the end of the sixth month. At the same time it undergoes counterclockwise rotation. Return to the original position occurred by the tenth postpartum day. The changes in position are accomplished with difficulty if as the result of previous inflammatory attacks the appendix has become adherent to some of the pelvic structures or is kinked or bent on itself. Under these circumstances the ground is prepared for an attack of obstructive appendicitis. Twyman and his associates on the other hand state that while they carried out no studies by X ray they did not observe marked displacement of the appendix at operation in any of their cases.

Of interest in this connection is the apparently unique case of torsion of the appendix in pregnancy reported by Flatley. In a review of the English literature he found only 1 case of appendiceal torsion reported by McFadden in 1926 and no instance of this accident associated with pregnancy in either the English or the Continental literature. His own case occurred in a 22 year old primipara 26 weeks pregnant whose symptoms of pain and vomiting with bilateral para umbilical tenderness more marked on the right had been present for 38 hours. Operation was undertaken on a diagnosis of red degeneration of an intramural fibroid. The appendix which was preileal was twisted through two and a half turns about half an inch from the proximal end. The distal portion was gangrenous. It unwound itself without mechanical aid. Premature labor occurred 11 days after operation.

The pathologic changes of appendicitis in pregnancy are probably not inherently more serious than at other times. They seem more serious however in almost every case because they are associated with another condition which although physiologic actually imposes a strain upon the entire maternal organism even in normal circumstances. The inflammatory process furthermore may actually be more severe in the pregnant than in the nonpregnant woman because it is often enhanced and aggravated by delay which for anatomic reasons has a more serious effect in the pregnant state.

Of 207 cases of acute appendicitis in pregnancy collected from the literature by Babler in 1908 almost 50 per cent were instances of perforated disease as were more than 64 per cent of the 28 cases which occurred during the puerperium. In the comparative series studied by Baer and his associates the ruptured and gangrenous types were respectively  $5\frac{1}{2}$  and  $3\frac{1}{2}$  times more frequent than in the nonpregnant state. In the 11 cases of appendicitis during labor collected from the

studied at the New Orleans Charity Hospital over a 15½ year period. Four occurred coincident with or immediately after abortion, 2 during labor and 3 during the puerperium. The remaining cases, 2 of which were fatal, occurred during gestation. There were 2 instances of pregnancy in the 158 fatal nonsurgical cases of acute appendicitis studied at Charity Hospital over the same period, and a third case in this group developed after an abortion which was probably criminal. High incidences are occasionally reported but for the most part as in these series the association of the two conditions is fractional.

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The increased vascularity and lymphatic dilatation which are physiologic during pregnancy predispose to the development of thrombosis and phlebitis and if abortion or labor should occur during or immediately after an attack of appendicitis the possibilities of their development are many times increased. The raw uterine cavity and gaping thrombosed sinuses of the pelvic vessels furnish ideal channels for the spread of bacteria and the risks of postabortal and postpartal infection may be added to those of acute appendicitis.

### CLINICAL PICTURE AND DIAGNOSIS

Although acute appendicitis is likely to be more serious late in pregnancy for the anatomic reasons stated the underlying reason for progression of the pathologic processes is exactly the same in the pregnant as in the nonpregnant state. *Diagnosis is delayed because it is difficult* treatment is not instituted because the diagnosis is not made and appendicitis in pregnancy as at any other time is a progressive disease whose gravity increases with the mere lapse of hours aside from any considerations of bacterial virulence or of individual resistance or lack of it.

The symptoms of acute appendicitis in pregnancy do not differ materially from the clinical syndrome at any other time but are more likely to be atypical because of the peculiar physiologic state. In addition the symptoms of the appendiceal disease overlap and are likely to be confused with the symptoms associated with the pregnancy particularly as the gestation advances.

In the first trimester of pregnancy morning sickness and the general malaise of early gestation may confuse the picture. In the second trimester as the uterus rises out of the pelvis right sided discomfort and vague abdominal pains are fairly frequent. As the pregnancy continues abdominal distention and discomfort flatulence constipation and similar symptoms develop and increase and it is a comparatively easy matter to belittle them in the belief that they are part of the discomfort which every pregnant woman expects to undergo or of which her obstetrician expects her to complain. Symptoms near term are likely to be considered as evidences of impending labor and symptoms during the puerperium may be regarded as due to puerperal infection. The following cases illustrate these points.

**Case 35** A white woman 6 months pregnant had sharp epigastric pain 60 hours before admission to the hospital. She had suffered from indigestion



literature by Norton and Connell rupture gangrene, or peritonitis was clearly present in at least 11. The 2 cases which occurred during labor in the Charity Hospital series were both instances of advanced disease. Royster, Jerlov, Meiling, Cosgrove and others all have emphasized the more serious character of appendicitis in pregnancy, particularly in the last trimester and during labor.

All of the circumstances like in pregnancy favor the spread of the disease process. The omentum and the loops of the intestine which would ordinarily assist in localization are crowded out of the way by the rising uterus which is now an abdominal rather than a pelvic organ. The uterus frequently changes its position, the changes being more frequent and more pronounced after quickening has occurred. The movements of the child become more vigorous the nearer term it approaches. If the attack occurs during labor, localization is scarcely to be expected because of the physiologic excursions of the uterus. Then as Marbury points out the movements of the organ have all the effect of a violent purgative. If localization by any chance has begun to occur it is likely to be destroyed by the abrupt and extensive alteration in the intra abdominal relations which is inevitable as soon as delivery has occurred.

A case in the Charity Hospital series illustrates the latter point.

*Case 34.* A 19 year old Negro woman was admitted at term with a vague history of labor pains for 24 hours preceded 48 hours earlier by occasional vomiting and nonradiating pain in both lower quadrants. She had been given a prescription for sleeping pills by her physician and had taken a double dose with the result that she was too stuporous to furnish any information. She delivered while in the observation room. She remained stuporous for another 24 hours during which time moderate abdominal distention was observed and no peristaltic sounds were elicited. Rectal examination revealed nothing abnormal and a diagnosis of adynamic ileus was made. When the effects of the sleeping pills wore off the abdomen was found to be extremely tender throughout and exploration was undertaken through a lower left paramedian incision on a diagnosis of probable intestinal obstruction confirmed by the roentgenologic demonstration of multiple fluid levels. The peritoneal cavity contained a large amount of purulent fluid. A localized abscess in the right lower quadrant contained what appeared to be feces. The appendix was large, gangrenous and perforated but was sealed off in the midportion; it lay directly over the ureter. Appendectomy was performed and sulfathiazole (10 gm.) was placed in the abscess cavity and in the wound. Parenteral sulfathiazole was also administered. Although a blood level of 90 mg. per cent was achieved there was no clinical response but recovery was progressive when penicillin was substituted.

Following an infusion the patient had a chill and shortly afterward she had an exacerbation of abdominal pain which then became generalized. The diagnosis of acute appendicitis was suggested by the interne on the service but was not entertained by the staff.

Conservative treatment was instituted including transfusion. On the fifth day the patient delivered a 5 month old dead fetus. The following day fluid was present in the chest and a massive pneumonia of the left lower lobe was noted. The patient was delirious. Sulfapyridine was administered without effect and death occurred on the eighth day. The diagnosis of acute appendicitis with generalized peritonitis which had been made just before death was confirmed by postmortem examination.

The atypical character of acute appendicitis in pregnancy and especially in (premature) labor is clear from this case. Conservative treatment which was carried out empirically was unsuccessful as it usually is in pregnancy. It should be noted that fetal death occurred in the absence of operation.

Pain is the most common symptom of appendicitis in pregnancy just as in the nonpregnant state but the location is apt to be higher than usual and the area of hyperalgesia is also likely to be higher. The movements of the child may increase the pain; an instance of this kind was observed at the New Orleans Charity Hospital and Marbury and Jerlov both noted this phenomenon. Nausea and vomiting may or may not be present just as in the nonpregnant state and the temperature elevation, acceleration of the pulse rate and similar findings show no differences in the pregnant woman. Since a moderate leukocytosis is physiologic in pregnancy a white blood cell count under 12 000 or 14 000 per cu. mm. is not of much significance though an increase in the percentage of polymorphonuclear leukocytes is frequent.

Examination is often attended with difficulties. Localized tenderness may be hard to elicit because of the size of the abdomen. The attenuation and thinning of the abdominal muscles which are notable in late pregnancy tend to diminish muscle spasm. Bimanual examination is not satisfactory in late pregnancy for the appendix and the adnexa are out of reach of the examining fingers. If the whole examination is conducted with the patient on her left side the heavy uterus will be at least partially removed from the field of investigation.

The history of previous attacks is the most valuable single point in making the diagnosis. When the pregnant woman's history is first taken she should be specifically questioned on this point and the reply should be incorporated in her record.

The differential diagnosis includes practically all of the conditions

throughout her pregnancy. Some hours after onset the pain localized in the right lower quadrant of the abdomen. She was treated by her physician with sodium bicarbonate and bromides and finally with two injections of morphine. She was somewhat relieved after two enemas had been taken. Fever of unknown degree had been present for several hours. The temperature on admission was 98.6° F, the respiratory rate 80 and the pulse rate 24. The patient was acutely ill. Physical examination revealed generalized abdominal tenderness, most marked in the right lower quadrant. The white blood cell count was 17,250 per cu. mm. with 90 per cent polymorphonuclear leukocytes.

Immediate appendectomy was carried out under ethylene ether anesthesia through a McBurney incision which was enlarged by the Weir technique. The appendix was gangrenous and a local peritonitis was present. The abdominal wall was drained.

Routine measures for peritonitis were instituted including transfusion and oxygen therapy. On the sixth postoperative day the patient's breath smelled of acetone; at this time the CO<sub>2</sub> combining power of the blood was 48 volumes per cent. A massive wound infection was treated by the Carrel-Dakin method. On the tenth day acetone was found in the urine. On the thirteenth day the patient was delivered of a dead fetus by low forceps; no heart sounds had been heard for several days. Pneumonia was diagnosed on the following day and death occurred on the fifteenth day. The antemortem white blood cell count was 58,500 per cu. mm. and there was a marked shift to the left. The antemortem temperature was 107° F. by rectum. Postmortem examination revealed streptococcic peritonitis, septicemia and terminal bilateral hypostatic pneumonia.

Diagnosis was confused in this case by the indigestion from which the patient had suffered throughout her pregnancy and by the two injections of morphine which masked the clinical manifestations. It should be noted that the fetus apparently died as the result of maternal toxemia; labor was not precipitated by operation.

*Case 36.* A white woman, 29 years of age, who believed herself to be 11 months pregnant, experienced a sharp pain about the umbilicus accompanied by nausea and vomiting 29 hours before she was hospitalized. Some hours after the onset the pain localized in the right side. Fetal movements had become progressively less since a fall 7 days before and no movements had been felt since the onset of the illness. The temperature was 98.6° F, the pulse rate 90 and the respiratory rate 20. The patient seemed moderately ill. Physical examination revealed bilateral lower abdominal tenderness but no rebound tenderness and no rigidity. The white blood cell count was 16,400 per cu. mm. with 96 per cent polymorphonuclear leukocytes. Blood chemical findings were within normal range.

The admission diagnosis was threatened premature labor versus pyelitis (which was excluded when urinalysis proved negative) versus salpingitis.

A 36 year old woman whose pregnancy had been uneventful had a dry labor terminated by section because of inertia. She had been given morphine and purgatives. Before operation the temperature was normal the pulse only slightly elevated and the general condition was regarded as fair. The first post operative day the abdomen was tympanic. The patient was given a purgative which was repeated the following day and prontosil was also used. Forty eight hours after the section the abdomen was re opened and a gangrenous appendix and large retrocecal abscess were found. The gangrene had extended to the cecum.

The appendix had not been seen at the first operation and it was postulated that the uterine inertia had actually been the result of spreading peritonitis though the grave findings were out of all proportion to the mild clinical picture even when the effect of morphine was taken into consideration.

The following case from the Charity Hospital series illustrates the difficulty of diagnosis during labor and the advantage of continuous observation.

*Case 37* A white primipara was admitted in apparently early labor with fleeting irregular pains. Thirty six hours after admission she complained of pain in the right side of the abdomen which was tender to palpation but not rigid. The temperature was normal. A few hours later pain was more marked in the right lower quadrant and there was tenderness on superficial palpation but no rigidity. Fifty two hours after admission the patient went into active labor with hard contractions every 3 minutes. Tenderness was now marked in the right upper quadrant. The white blood cells numbered 14 500 per cu mm with 82 per cent polymorphonuclear leukocytes. The original diagnosis of possible ureteral calculus having been ruled out by negative roentgenologic examination exploration was undertaken on the diagnosis of acute appendicitis after the delivery of a 5 pound 12 ounce boy by low cesarean section. The appendix was retrocecal and gangrenous but not ruptured though the wall was as thin as tissue paper over the several fecaliths contained in the lumen. Appendectomy was performed and recovery was smooth the postoperative regimen included sulfathiazole therapy.

#### MANAGEMENT

Some surgeons and some obstetricians are so impressed with the possible seriousness of acute appendicitis in pregnancy that they have suggested either prophylactic appendectomy in married women who have had repeated attacks of appendicitis or appendectomy as soon as pregnancy is diagnosed before another attack occurs. The wisdom of interval appendectomy in a patient subject to repeated attacks of ap

discussed in the chapter on differential diagnosis (p 155) All the routine diagnostic procedures should be carried out No great amount of time should be spent in excluding such conditions as intestinal obstruction mesenteric thrombosis, and strangulation of uterine and ovarian tumors or of the pregnant uterus itself All of them are surgical states some of them are extremely urgent and if the abdomen should be opened on an incorrect diagnosis the patient would be benefited by the error

*Operation would naturally be undesirable in any purely medical state or in gynecologic conditions such as bilateral or right sided salpingitis even though they might eventually require operation Pyelitis is often very difficult to exclude It is relatively frequent in pregnancy for anatomic reasons and it occurs about six times more often on the right side than on the left side The pregnant uterus usually rotates to the right and therefore may compress the ureter as the latter crosses the pelvic brim The presence of pyelitis naturally does not exclude acute appendicitis for the coincidence though unusual is perfectly possible*

At or near term and during labor all the usual conditions must be excluded as well as such obstetric conditions as eclampsia placenta previa abruptio placentae and rupture of the uterus The course of events in the first three conditions usually makes the diagnosis clear within a brief time Rupture of the uterus may be more confusing as illustrated by the case reported by Louyot and his associates

Low cesarean section was undertaken in a 20 year old primipara after a prolonged labor with hard pains and no engagement Subtotal hysterectomy was performed after a rupture of the lower segment was found The patient complained violently and went into shock when the uterus was exteriorized She improved only slightly after transfusion and other routine measures From the third postoperative day she exhibited increasingly clear signs of peritonitis and death occurred on the seventh day Postmortem examination revealed a ruptured appendix and generalized peritonitis the operative site was in good condition

Hamant in discussing this contribution pointed out that if autopsy had not been carried out the involvement of the appendix would never have been suspected and the death would have been charged to the obstetric procedure

The difficulty of diagnosis when acute appendicitis occurs in labor is apparent in all reported cases beginning with the original report by Grattan in 1919 Quin's case is a particularly good illustration

enhanced if labor follows promptly on operation and which is still greater if drainage is necessary. The risk of evisceration and the less serious possibility of ventral hernia however must be taken in view of the great risk to life inherent in the circumstances.

*The Obstetric Factor* Unless the patient is at term<sup>1</sup> or actually in labor the obstetric factor may be disregarded. She is a surgical subject first and the wisest and safest plan in the absence of obstetric indications for an operative delivery is to let nature take care of the obstetric aspect of the case. Unless the patient is fairly near term labor is not likely to be precipitated by the operation and even near term the pregnancy is not disturbed in a surprising number of cases.

If labor should be precipitated the patient should be permitted to deliver by the natural channel whenever that is possible. If the abdominal closure has been done carefully and if parturition is shortened by the various methods known to modern obstetric art the end result is likely to be more satisfactory than the result of any procedure that involves the termination of pregnancy at the time of operation. If a real contra indication to vaginal delivery exists such as an absolutely contracted pelvis there is no other choice than abdominal section preferably low section which is best carried out at the time of the appendectomy if the patient is close to term. Although Porro section is a regrettable operation in a young woman the patient's best interests are often served by it if rupture or abscess formation has occurred and if delivery by the natural route is impossible. If labor has actually supervened as in the case just recorded from the New Orleans Charity Hospital there seems no alternative to combined cesarean section and appendectomy undesirable as is the combination. In this case packing off of the operative field was done with the greatest care.

Morphine should be used liberally for the first few days after operation in an endeavor to prevent abortion or premature labor. Purgatives laxatives and enemas should be withheld and distention should be controlled by constant intestinal decompression and the rectal tube. Lackner and Tulsky have suggested the use of progesterone in the postoperative routine because of its demonstrated inhibiting effect on the motility of the human puerperal uterus and its clinical results in habitual abortion. If sulfanilamide or its derivatives are used the possible effect on the liver and kidneys must be borne in mind because of the strain to which these organs are already subjected as the result of the pregnancy. Penicillin in large doses is preferable. Otherwise the postoperative routine does not differ from that after the usual operation for appendicitis.

pendicitis does not seem open to argument. Whether prophylactic appendectomy should be performed early in pregnancy is somewhat doubtful though it seems logical to advise surgery if an attack supervenes during pregnancy especially during the first trimester to eliminate the risks of more serious consequences when gestation is farther advanced. In other words there seems no reason why acute appendicitis should not be handled in the pregnant woman exactly as in the non-pregnant that is by immediate operation as soon as the diagnosis is established or by exploration if the diagnosis cannot be positively excluded.

The majority of writers who advocate conservative treatment for appendiceal peritonitis and abscess believe that it has no place in the management of appendicitis in the pregnant woman because of the impossibility already pointed out of securing the state of physiologic intra-abdominal rest which is an essential part of the treatment. The farther the gestation has advanced the more do conditions within the abdomen militate against localization. It is best therefore to perform appendectomy at once if the appendix can be removed safely and expeditiously. Otherwise drainage can be instituted though other things being equal mere drainage should be avoided whenever possible. If it must be used the tubes should be placed with great care for changing conditions within the abdomen particularly if abortion or labor supervenes are likely to disturb their position. All manipulations should be reduced to a minimum and the gravid uterus should be handled as little as possible and under no circumstances should be brought out of the wound. As Kelly and Hurdon note the entire operative procedure should be conducted on the assumption that infection is present and is likely to spread.

Anesthesia and incision are both determined by the desires of the surgeon though local analgesia is to be avoided. Some authorities advocate the McBurney incision others the right rectus but it makes little difference which is used if the surgeon remembers that the appendix is higher in the abdomen than it usually is and if he places the incision accordingly. Either the right rectus or the Battle incision seems logical however because of the difficulties of surgical access in the pregnant woman and the likelihood that the appendix because of the space occupied by the enlarged uterus will be in the lateral recesses of the abdomen.

Closure should be done with special care and more sutures than usual (preferably nonabsorbable) should be used for the continuation of pregnancy will put a heavy strain on the operative scar which is

moribund when the fetus is expelled They abort because they are dying They do not die because they have aborted

The high fetal mortality aside from the loss of life by abortion is partly due to prematurity and partly to the effect of the maternal fever toxemia and other constitutional disturbances on the child

### CONCLUSIONS

1 The general principle upon which the management of acute appendicitis in pregnancy should be conducted is that the association of the two states does not in any way alter either the risks associated with the surgical disease or the surgical principles upon which its treatment should be based Acute appendicitis is always acute appendicitis

2 The point of view of the surgeon and that of the obstetrician do not conflict and the patient's interests are best served when both physicians work in close co operation if for no other reason than to prevent the narrow point of view which each one might exhibit if he handled the case alone

3 Pregnancy although a physiologic state is nonetheless associated with certain risks There is no reason therefore for subjecting the pregnant woman to an additional risk by letting a potentially fatal disease run its course without taking the same steps to control it which would be taken if she were not pregnant The only difference between acute appendicitis in the pregnant and in the nonpregnant state is that in the former two lives are jeopardized instead of one A pregnant woman with acute appendicitis is in a precarious situation and her risk is increased by her pregnancy The status of her pregnancy is not improved by any delay in handling her surgical condition but rather is further imperiled Therefore the treatment of acute appendicitis in pregnancy just as at any other time should be exclusively surgical



## MATERNAL AND FETAL MORTALITY

Theoretically the mortality of acute appendicitis in pregnancy should be no higher than in the nonpregnant state and in the first trimester it usually is not. Thereafter the symptoms and signs of appendicitis become more and more modified by the circumstances of the pregnancy, diagnosis becomes more difficult, treatment is instituted late, the pathologic process is correspondingly advanced, and the mortality reflects these facts. In 274 cases collected by McDonald, including 204 cases previously collected by Jerlov, the mortality for intrinsic disease was 0.71 per cent, for abscess 23.5 per cent, and for peritonitis 30 per cent. Smith and Bartlett reported 3 deaths in 20 cases. Marbury reported a mortality of 26.4 per cent in 34 collected cases occurring in the last trimester. Over a 15½ year period at the New Orleans Charity Hospital there were 2 deaths in 23 surgical cases and 3 additional nonsurgical deaths. There is no reason to believe that the situation is any better generally than these statistics would indicate.

The incidence of abortion and premature labor after appendectomy is high, but both accidents can more readily be explained by the disease process and by delay in surgical treatment than by the operation itself. It is equally high if no operation is performed. In 89 cases of acute appendicitis with perforation during pregnancy collected by Babler, operation was done in 80, abortion occurred after operation in 37 cases, it is true, but it occurred before operation in 33, and it also occurred in 9 of 13 cases with perforation in which no operation was done. Most of the operations seem to have been performed late, since the maternal mortality was 45 per cent.

The frequency of abortion in later series varies but is usually roughly proportionate to the seriousness of the disease. Jerlov reported an incidence of 13.8 per cent in intrinsic disease, 55 per cent in abscess, and 63 per cent in peritonitis. In McDonald's comparable study the figures were respectively 11.4 per cent, 66 per cent, and 72 per cent. Abortion or premature labor occurred in 8 of the 16 cases reported by Smith and Bartlett, the accident occurring in 4 of 5 cases in which drainage was carried out and in 4 of the 11 cases in which it was not required.

The mortality is highest among women who abort, but the part of abortion in the outcome is likely to be overestimated or wrongly evaluated. Abortion is not the primary cause of the fatality. Many women are overwhelmed by toxemia and sepsis and are actually

22 April 1939 the case was reported of a white woman 19 years of age who while at work fell off the stool on which she was standing and struck her right side against the sharp edge of a metal table. She was not seen to fall but 15 minutes later described the accident to her fellow employees and was seen holding her side on which a swollen bruised area was present. At operation 19 days later the appendix and cecum were acutely inflamed and the patient died shortly afterward from peritonitis and pneumonia. Her family was granted compensation by the local court and when the employer appealed the Michigan Supreme Court upheld the award on the basis of the physician's testimony in regard to the possible chain of events.

Although in this case judgment was rendered for the plaintiff the time elapsed in the opinion of many observers would be an argument against the traumatic origin of the appendicitis. On the other hand Goodyear reported the case of a 37 year old WPA worker without previous gastro intestinal disturbances who while at work slipped and fell so that his abdomen was wedged between the rims of two sewer mains each weighing 300 pounds. When he was released he immediately complained of pain and shortly afterward began to vomit persistently. At operation 36 hours later a retrocecal ruptured appendix was found with a spreading peritonitis. A stormy postoperative course terminated in recovery. Though the cause and effect relationship seems perfectly clear compensation was not granted in this case because the WPA Commission in Washington held that there is no such entity as traumatic appendicitis.

#### INCIDENCE

At best the incidence of authentic cases of traumatic appendicitis is small. Surgeons of large experience such as the late John B. Deaver never saw a case. In 1905 Kelly and Hurdon made the statement that the disease is commoner than it seems to be and is simply overlooked because the history seldom includes inquiries about preceding trauma. Connell who questioned 87 surgeons about their views on the subject found that although only 24 had personally seen instances of traumatic appendicitis 31 felt that trauma could be an etiologic factor in acute appendicitis 6 that trauma might initiate an attack in a previously diseased appendix and 11 that strain might play a part in the causation of the disease. Fowler who analyzed 243 opinions found that 24 per cent indicated a possible primary and 56 per cent a possible secondary relationship.

## ( XVI )

### *Special Types of Acute Appendicitis*

#### IV TRAUMATIC APPENDICITIS

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##### MEDICO-LEGAL ASPECTS

Although the number of cases of supposed traumatic appendicitis is small and the number of proved cases smaller the subject is still worth special consideration because of its medico legal aspects. Industrial surgeons, insurance companies and courts have acknowledged the existence of this type of appendicitis and payments have been made for it just as for any other industrial accident though this policy for obvious reasons is by no means uniform. Aside from the difficulty of proving that acute appendicitis in a given instance is actually due to trauma from the insurance standpoint this is a condition which frequently puts the policyholder on the horns of a dilemma. Accident insurance policies specifically exclude claims for illnesses and sickness policies specifically exclude claims for accidents which often means that the holder of one kind of policy or the other or even of both kinds may have difficulty in proving his claim.

In 48 cases of traumatic appendicitis collected from the literature by Fowler 24 verdicts were rendered for the defendant and 24 for the plaintiff. In 6 cases in which appeals were taken 4 decisions (3 for the petitioner and 1 for the defendant) were reversed and 2 (1 for the petitioner and 1 for the defendant) were sustained. In several cases claims were allowed by the insurance companies without argument. In 8 death claims damages were recovered in all but 1. In 8 cases observed by Fowler and apparently not included in the collected series the claims were dropped in 3 cases and settled in favor of the defendant in 1. In the remaining cases judgment was rendered for the plaintiff in 1 instance in the sum of \$11 000. According to Fowler's calculations the courts favor the plaintiff 51 in cases of direct injury but only 21 in cases of indirect injury.

There still seems no uniformity of action by the courts however. In the legal section of the *Journal of the American Medical Association* for

## PATHOLOGIC PROCESS

In addition to its medico legal aspects traumatic appendicitis is worthy of special consideration because it is frequently of a very serious type. Although not all of Kelly and Hurdon's 50 collected cases can be accepted at their face value it is significant that in 37 the appendix was either perforated or gangrenous. Other observers report approximately the same proportions of complicated cases. The appendix was perforated or gangrenous or both in all 4 cases reported by Bissell for instance in all 3 reported by Ray and in 4 of the 9 personal cases reported by Fowler. Delay in diagnosis and treatment due to confusion in the history might account for the advanced pathologic process in some of these cases but in others the chain of events progressed very rapidly. Thus in a case reported by Ray a child kicked in the abdomen had immediate pain tenderness nausea and vomiting and a gangrenous appendix was found at operation 18 hours later.

External evidences of injury have been reported in only a few instances and possible evidences of trauma in the appendix are usually obliterated by superimposed lesions. Fowler however was able to collect 23 cases in which the appendix presented traumatic lesions including in addition to perforation laceration hemorrhagic suffusion local ecchymosis avulsions of the serosa that sometimes involved adjacent structures hematoma and vascular injury. His point is well taken that the appendix should be preserved with particular care in all cases of supposed traumatic appendicitis because many of the specimens will eventually have medico legal value.

## MECHANISM OF INJURY

While the appendix is usually one of the best protected and most deeply lying of the abdominal organs there are numerous exceptions. It is often found hanging over the pelvic brim or lying directly against the abdominal wall. It may be relatively superficial in thin persons or in persons with deficient abdominal musculature. Furthermore even when the appendix is deeply located and fully mobile the abdomen is not a solid mass in which force can act only in the direction in which it is applied. It is a liquid and gaseous mass and force exerted against it is transmitted in all directions (Ludington). As Fowler pointed out if direct force is transmitted to gaseous fluid semifluid and solid media beneath the abdominal wall such media are subject to displacement.

The 50 instances collected from the literature by Kelly and Hurdon in 1905 cannot all be accepted as authentic. Very doubtful is the case which developed at night after a day of hunting or that of the youth who ran over at 12 years of age had recurrent attacks of appendicitis for the next 8 years. Kessler found no instance in 1000 personally studied cases and Ry only 3 instances in 1500 cases, 1183 of which were acute disease. Jennings and his associates found only 11 instances in 1680 cases of acute appendicitis all occurred in thin wiry individuals under 20 years of age. There were only 29 possible (not proved) instances of traumatic appendicitis among the 6441 surgical cases of acute appendicitis studied at the New Orleans Charity Hospital over a 15½ year period. 3 were fatal. Thirteen, 1 of which was fatal, occurred in children under 13 years of age and 15, 1 of which was fatal, occurred in the age group between 13 and 39 years of age inclusive. The only case in a patient over 40 years of age terminated fatally and is summarized herewith.

*Case 36.* A white male, 62 years of age, was hospitalized for a fracture of the neck of the left femur for which a Thomas splint was applied. The injury had occurred when the patient fell with another man on top of him. On the tenth day of hospitalization extreme abdominal distention developed and large quantities of yellow fluid were obtained when Wangensteen suction was instituted. The temperature was 101.2° F, the pulse rate 110 and the respiratory rate 30. The interne suggested that the ileus which was obviously present might be due to a ruptured appendix in spite of the absence of symptoms referable to that organ and postmortem examination when the patient died on the fourteenth day of hospitalization confirmed the suspicion.

In this case the lack of abdominal pain and of other symptoms and signs referable to the appendix is notable and is not infrequent in patients advanced in years. It is possible that this case is an instance of traumatic appendicitis though there was a long lapse of time between the injury and the first *clear cut* sign of abdominal pathology.

Royster furnished an excellent history of the type of case which probably is often reported as an instance of traumatic appendicitis but which really is not. A football player the night before a game apparently had an attack of ptomaine poisoning. The following day he entered the game, did not play up to his usual form and finally had to leave the field. A few hours later he was operated on for acute appendicitis which probably would have been considered of traumatic origin except for the more reasonable explanation that his food poisoning of the night before was really the onset of acute appendiceal disease.

precipitate an attack of acute appendicitis Burgess reported a case which substantiates this reasoning

A 46 year old Hawaiian was in the habit of using a pneumatic drill suspended overhead the butt end of which he rested in the right iliac fossa so that he could exert pressure on the tool Two weeks after he had begun to use the drill while at work he had a sudden disabling pain in the right iliac fossa When he felt sufficiently relieved to resume drilling he found that he could not rest the tool in its former position For the next 24 hours the pain was dull and continuous then he had a sudden exacerbation and operation was performed The appendix which lay to the right of the cecum was directly over the psoas muscle was sharply kinked upward at the base and was tightly bound to the posterior parietal peritoneum by old tough adhesions extending from the kink The tip had been amputated

Compensation was granted without question in this case

#### CLINICAL PICTURE AND CRITERIA OF DIAGNOSIS

The symptoms of traumatic appendicitis do not differ from those of the usual case of appendicitis except that the abdominal pain is often of greater severity With this exception the majority of cases of traumatic appendicitis seem to present the classical picture of acute disease and the frequent delay in diagnosis is due not to lack of symptoms but to the confusion caused by the history of trauma In many of the reported cases apparent subsidence of the symptoms caused by the injury was followed after a time by renewed symptoms indicative of rupture and peritonitis This type of appendicitis is apparently most frequent in children of both sexes and in men

A most unusual case was reported by Mortenson and Bray

A nursemaid 14 days before she was first seen lifted a heavy basket and immediately complained of right sided pain followed by vomiting Both symptoms continued for 3 days after which persistent right sided soreness was felt Nine days later the girl was kicked in the right side by the child she was nursing Operation 36 hours later revealed the proximal half of the appendix acutely inflamed probably the authors surmised as the result of the more recent injury while the distal half was gangrenous and lay in an abscess cavity probably as the result of torsion caused by the first injury which had been followed by the development of true obstructive appendicitis

In a case observed at the New Orleans Charity Hospital the history of trauma was complicated by the existence of pelvic inflammatory disease and uterine fibroids

and to pressure changes while fixed structures are subject to particularly great stress

Possible causes of direct injury to the appendix do not differ from the causes of other abdominal injuries. They include automobile accidents in which the patient is run over crushing injuries falls kicks and blows. Indirect causes such as strains due to lifting heavy objects prolonged exertion and falls and other injuries which do not directly affect the abdomen are decidedly less convincing. Ludington reported a case in which conscientious deep palpation through a thin abdominal wall during a probable attack of acute appendicitis undoubtedly precipitated the rupture observed shortly afterward at operation.

If direct evidence of injury is not present in the appendix it is necessary to postulate the chain of events. The most obvious and probably the correct explanation is overdistention of the lumen of the appendix by a sudden influx of fecal contents as the result of trauma. The high percentage of fecaliths reported in cases of traumatic appendicitis supports this point of view. They were present in 30 of the 50 cases collected by Kelly and Hurdon and in 65 per cent of Brunigs cases (cited by Fowler). Although most series unfortunately are less specific on this point. In an appendix containing a fecalith an influx of fecal material may conceivably cause impaction and the chain of events described under the heading of obstructive appendicitis (p. 135) may thus be set up. Angulations bends adhesions, strictures and similar predisposing causes would operate just as in other cases of obstructive appendicitis. It is also reasonable to assume that trauma may cause minute breaks in the mucosal coat which would permit invasion by bacteria.

Gretve who granted that trauma may be followed by inflammation of the appendix doubted that the disease should be regarded as true appendicitis even if necrosis ensues. On the other hand he considered it at least theoretically possible that direct violence to the right iliac fossa may become the exciting factor in the production of appendicitis in an organ that is the seat of old pathologic changes. He doubted that in practice it is ever possible to exclude with certainty the possibility that the appendix before the accident had been the seat of a quiescent or developing inflammatory process that up to that time had produced no manifest symptoms.

Byron Robinson (cited by Fowler) contended that if the appendix lies on the psoas muscle normal muscular contractions may irritate it and that acute flexion of the thigh on the trunk as in bicycling might

least temporary cessation from work the nature and location of the injury are such as to affect the appendix and the symptoms of appendicitis directly follow the injury

5 In case of death autopsy must show that the appendix because of an abnormal position and condition could have been affected by the injury

### THERAPY

The treatment of traumatic appendicitis like the treatment of any other variety is immediate operation In view of the serious nature of the pathologic process often found in such cases delay is even more unwise than usual and if the surgeon cannot exclude appendicitis promptly he would be well advised to resort to operation at once



**Case 39** A colored woman 36 years of age developed abdominal pain an hour after she had been set upon and beaten by several men 5 days before her admission to the hospital Two days later she had a profuse vaginal discharge with burning on urination and fever of unknown degree Nausea and vomiting had been present since the onset of the pain The temperature was 99° F the pulse rate 90 and the respiratory rate 20 Physical examination revealed marked upper abdominal rigidity, less marked rigidity in both lower quadrants and pain and rebound tenderness on pressure in the left iliac fossa Pelvic examination revealed uterine fibroids

The white blood cell count was 33 900 per cu mm with 86 per cent polymorphonuclear leukocytes The sedimentation rate was 26 minutes The urine contained 2 plus albumin pus and a few hyaline and granular casts

The patient was admitted to a gynecologic service with a diagnosis of traumatic abdomen uterine fibroids and acute pelvic inflammatory disease Nine days later when bed rest and supportive measures had resulted in no improvement a surgical consultant suggested the diagnosis of kidney injury Cystoscopy and blood chemical examinations revealed nothing abnormal The temperature at this time was 103.8° F and the pulse rate 120 The following day a phlegmon in the right iliac region was incised under spinal analgesia but no pus was found Its origin was not determined The patient died 3 days later Postmortem examination revealed a ruptured appendix generalized peritonitis and a subphrenic abscess The evidence could readily be traced upward from the peritoneal cavity to the subphrenic space

This case is a possible instance of traumatic appendicitis The diagnosis was confused by the obvious pelvic inflammatory disease The existence of a subphrenic abscess was not suspected during life

The argument for traumatic appendicitis is necessarily of the *post hoc ergo propter hoc* variety and its logic as has been intimated is often open to question Clearly the most important consideration is that the symptoms must follow close upon the injury certainly within the space of a few hours while if operation is delayed there must be no doubt of the patient's continued illness with symptoms referable to the injury in the interim The following criteria have been outlined by Kessler Director of the New Jersey Rehabilitation Clinic

- 1 The individual before the accident must have been well and able to work and must have had no signs or symptoms of acute appendicitis
- 2 Severe injury affecting the abdomen or strenuous overexertion must be established
- 3 There must be immediate evidence of illness so severe that the patient is unable to continue work
- 4 The relationship between trauma and aggravation of pre-existent appendicitis can be accepted only when the injury is severe enough to require it

the collection of ailments he became afflicted with them all himself. In view of such bewildering and unrelated possibilities a diagnosis of chronic appendicitis is necessarily based largely on speculation and curiously an extra appendicular origin is frequently claimed for the syndrome which appendectomy is supposed to relieve.

Supposed to relieve ■ ■ fair enough comment. Large series of cases are on record in which appendectomy for so called chronic appendicitis not only did not improve the condition but many times made it rather the worse. Every conscientious surgeon can supply similar instances from his own experience. One of the latest of these series and in many respects one of the most comprehensive was reported by Alvarez from the Mayo Clinic. His study of 385 patients which he asserted frankly brought him several surprises ■ worth citing in considerable detail.

The first of the surprises had to do with the indications for the appendectomies which had been performed. Only 130 of the 385 patients had histories of recurrent attacks or complaints of abdominal pain. In the remaining 255 cases the indications for operation included such diverse complaints (which are strongly reminiscent of Hertzler's collected list) as neurosis, fatigue states, mucous colitis, constipation, diarrhea, migraine, duodenal ulcer, cholecystitis, food allergy, renal colic, pyelitis and polyglandular dysfunction. In several instances the operation had apparently been performed merely because the patients insisted on it. In a large number of cases as might have been expected later and more careful study proved the original trouble to have been due to causes unconnected with the appendix including in 1 tragic instance carcinoma of the ascending colon. Incidentally I am personally aware of 2 instances in which appendectomy through button hole incisions was carried out on patients with carcinoma of the stomach and of 11 similar instances observed by Ochsner.

In most of the 255 instances in Alvarez's series in which appendectomy had been performed on obscure indications or none at all the original trouble persisted. That was why the patients were in his office. Two had had permanent relief, 4 temporary relief and 4 partial relief. The remainder had had no relief at all. Those figures mean Alvarez estimated that patients submitted to appendectomy on doubtful indications have 1 chance in 100 of obtaining permanent relief and 3 chances in 100 of obtaining partial or temporary relief from their complaints. The margin is too slim to warrant the risk inherent in any anesthetic however skillfully given and any laparotomy however skillfully performed. Furthermore many of the patients were worse after operation than they had been before, 34 of them being so much worse

## (XVII)

### *Chronic and Recurrent Appendicitis In Relation to Acute Appendicitis*

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The discussion of appendiceal disease that is not acute is no part of the purpose of this book. On the other hand the consideration of the acute disease will be clarified if the nonacute type is briefly considered. The chief purpose of such a discussion is (1) to distinguish between so called chronic appendicitis and the type of appendicitis characterized by recurrent acute attacks and (2) to eliminate certain misconceptions concerning nonacute disease which are very general and which therefore form the basis for rather general erroneous practices.

#### SO CALLED CHRONIC APPENDICITIS

Whether or not so called chronic appendicitis is a true clinical entity has been the subject of acrimonious debate. Hertzler who crusaded against the concept for many years of his life was perfectly positive that such a disease does not even exist. A trained pathologist himself he questioned the supposed pathologic findings in chronic appendicitis stating that there is no change found in appendices removed for supposed chronic disease which cannot also be found in appendices removed merely because the abdomen has been opened for some other reason. Boyd supported Hertzler's point of view by stating that the fibrotic and other changes supposed to be indicative of chronic appendicitis are wholly inadequate to explain the symptoms ascribed to them.

As Boyd implied the situation in regard to chronic appendicitis is even more confused clinically than it is pathologically. The syndrome is entirely inconstant. The chief symptom is discomfort or pain in the right side but it is not present in all cases and it is associated with a myriad of other complaints including gastric and gastro intestinal symptoms, menstrual irregularities, nervous disturbances, arthritis, neuritis, melan cholia, epilepsy, migraine, fatigue states, duodenal ulcer, cholecystitis and even fallen arches. Hertzler who collected this remarkable list from the literature disposed of it with the statement that as he contemplated

previous acute attacks (fig 73 p 342) In the 157 patients with acute appendicitis who died without operation during the same period there was a history of previous attacks in 18 In other words 11.7 per cent of these 477 fatalities were even more unnecessary than are the usual deaths in acute appendicitis for the victims had had ample warning before their final bells tolled

A few observers such as Lamb believe that previous attacks definitely increase the severity and speed the spread of the disease in subsequent attacks and so make rupture more likely Most observers take the opposite point of view which the Charity Hospital figures seem to justify the mortality of 3.16 per cent in surgical patients with previous attacks in this series is to be compared with the mortality of 6.02 per cent in the patients who had no previous history of appendicitis (fig 73 p 342) Part of the explanation may be that the patient is safer in a subsequent attack than in his first because having had his warning he will send for the physician promptly and will agree to operation more willingly The reverse however as all physicians know may also be true The patient because he has safely passed through previous attacks without operation may for that reason have acquired an unwarranted sense of security

### THE SURGERY OF RECURRENT ACUTE APPENDICITIS

Discussions of whether or not patients are safer actually or presumptively in subsequent attacks of acute appendicitis than in their first attacks are after all beside the point The crux of the matter is that no physician can foretell what is going to happen in or after any given attack He certainly cannot say that the attack in question will be the last the patient will ever have as it quite possibly may be or that it will be followed by another next week or next month as it quite possibly may be He certainly cannot say because the disease has not gone on to gangrene and perforation in the current attack that it will not go on to these dangerous stages in the next attack or even for that matter in the present attack The patient does not have *recurrent* acute appendicitis it should be remembered until he has recovered from the present attack

The physician's duty therefore given a patient with an undoubted history of attacks of acute appendicitis—the real disease and not its hybrid imitation—is to advise interval appendectomy If the patient does not take his advice it is his further duty to warn him of the risk of placing himself beyond surgical aid in the event of another attack

that they had submitted to from one to five other unprofitable operations in all sixty eight secondary operations were performed on this unhappy group

Appendectomy is a form of psychotherapy. Alvarez therefore concluded is utterly useless. If operation is advised at all on inadequate indications it should be advised frankly as an exploration (and should be carried out through an incision large enough to permit exploration of the entire peritoneal cavity) and not as an appendectomy and the patient should be told the whole truth. If then wrote Alvarez with the unfavorable odds before him an individual should choose to gamble and should win he couldn't logically heap abuse on the sensible clinician who cautioned him and heap praise on the highly optimistic surgeon who operated. He could only congratulate himself on being lucky. Those are sound comments on the status of appendectomy for so called chronic appendicitis.

In the remaining 130 patients in Alvarez's series all of whom had had one or more definite attacks of acute appendicitis the results of operation were quite different. Most of these patients had consulted him for new complaints. He inquired why appendectomy had been done merely because he saw the old scar. In this group 67 per cent of the patients were permanently cured. 25 per cent were partially or temporarily helped and only 5 per cent were no better and only 2 per cent were worse than before operation. When the story of previous acute attacks was unquestionable the percentage of cures ranged from 71 to 78 per cent. When the story was doubtful however only 48 per cent of the patients were permanently relieved.

### RECURRENT APPENDICITIS

Alvarez's illuminating report leads logically to the discussion of the type of nonacute appendicitis that is really appendiceal in origin that is the recurrent variety as contrasted with the type that is called appendicitis apparently in desperation and because there seems nothing else to call it.

All studies of acute appendicitis show a large proportion of patients who have had previous attacks. The number would undoubtedly be very much larger if the histories were properly taken. Unfortunately too all series show in this group of cases a certain proportion of fatalities. In 6441 cases of acute appendicitis studied at the Charity Hospital of Louisiana at New Orleans over an approximately 15½ year period there were 320 deaths. 1758 patients of whom 38 died gave a history of

## ( XVIII )

### *Factors of Mortality in Acute Appendicitis*

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In 1939 speaking before the American College of Physicians Reginald Fitz distinguished son of the physician to whom we owe the modern concept of appendicitis made a statement which came from him with peculiar force because of his heritage Appendicitis he said in spite of being a fashionable and well studied disease for more than 50 years continues to slap our faces insultingly A quarter of a century before John B Murphy had said almost the same thing The hospital mortality of appendicitis should be a blow in the solar plexus of our surgical conceit In spite of the steadily improved mortality of acute appendicitis in the last several years (fig 76) both of those remarks are still applicable

Throughout this book there has been a constant endeavor to correlate the end results of acute appendicitis particularly the fatal end results with the clinical and therapeutic circumstances in which these results occur It is abundantly clear both statistically and otherwise why patients die from this disease Nevertheless it seems of value to review the possible factors of mortality in acute appendicitis because when they are brought together under a single heading they are likely to be more impressive than when they are considered singly and because it will thus be evident that in this enlightened medical day many if not most of these factors should no longer play any considerable part

Spreading peritonitis with its complications is the immediate and overwhelming cause of most deaths in acute appendicitis Reports in the literature credit it with the responsibility for from 70 to 95 per cent of the mortality in this disease In the Charity Hospital surgical series it was directly responsible for 215 of the 320 deaths and its complications were responsible for at least 47 others In the nonsurgical series of 158 fatal cases peritonitis was responsible for 145 of the deaths

Deaths from pneumoma cardiac disease and other complications have been discussed in detail and need no further comment though attention might be called to 8 fatal cases in the series 6 in the age group 13-39 years in which the fatality could not be explained even in the 2

He should warn him against long sea trips against business trips to remote places and against hunting camping and fishing expeditions that would put him out of reach of a surgeon. Rescues of patients with acute appendicitis from such predicaments are rather frequent and are always dramatic but the outcome is not always happy and the risk is always ill advised. The law of averages may be against the recurrence of the disease under the circumstances but the law of averages never protected any individual against anything. Similar advice should also be issued to the patient who has had previous conservative treatment for appendiceal disease that has gone on to rupture and abscess formation or who has had surgical treatment that did not include removal of the appendix (p 208).

On the other hand it should be emphasized again that even the simplest operation is attended with risks inherent in the anesthesia and in the procedure however carefully both may be carried out. Collected statistics show mortalities for nonacute appendicitis ranging from fractions of 1 per cent to 2 per cent and more. No series of major operations—and appendectomy even for nonacute disease is a major surgical procedure—can be carried out over long periods of time without a certain inevitable proportion of deaths from unexpected causes or from accidents and technical errors. Respiratory complications unexplained infections embolism even peritonitis may develop in the most unlikely cases. Unnecessary operations should therefore be guarded against particularly on insufficient indications while at the same time surgery should be advised in the interval for the patient with recurrent acute appendicitis who as has already been pointed out does not have the recurrent variety of disease until he has safely passed out of the particular attack in which he currently finds himself.

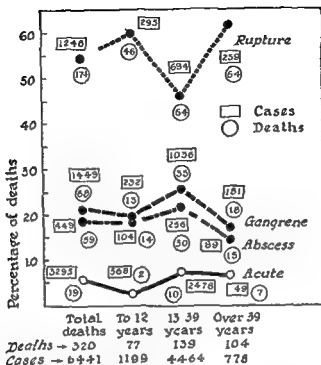


Fig 77 Proportionate distribution of deaths in relation to age and pathologic process in 6 441 surgical cases of acute appendicitis

### STATISTICAL ERROR

Attention has been directed in the preface of this book to the illogic of reporting as a single group cases of chronic and recurrent appendicitis and of acute appendicitis. From the psychologic standpoint if no other this is very bad practice. It gives an entirely false idea of the true status of the acute disease and is unquestionably at least partially responsible for the complacency with which both professional and lay persons are prone to regard acute appendicitis.

Almost invariably when series consisting of both acute cases and recurrent or chronic cases of appendicitis are broken down it is found that the mortality for interval appendectomy is fractional or even nil and that the chief or the whole mortality is concentrated in the acute group. When the figures are combined the mortality for the whole series is brilliantly and misleadingly low. It is unfortunate that the figures for the Bureau of Census are thus made up. It might be well as an occasional writer has suggested to make up the statistics of acute ap



cases in which postmortem was carried out. Most of the patients died rather suddenly though the clinical picture was not suggestive of embolism. In the occasional case as in 1 instance of cerebral hemorrhage and 1 of suicide death was due to causes unrelated to the disease but with these few exceptions all of the patients who died in this series as in any other series of cases of acute appendicitis died of that disease and its complications and nothing is gained by not facing that fact frankly.

Death rate (number per 100 000 population)

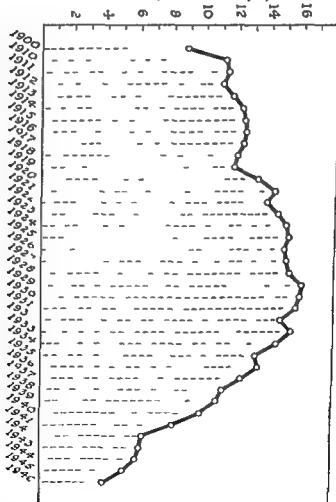


Fig 76 Death rates for acute appendicitis per 100 000 estimated population in registration states for years 1900 and 1910-1946. Figures are from the National Office of Vital Statistics, Washington, D. C. The rate for 1946 is estimated.

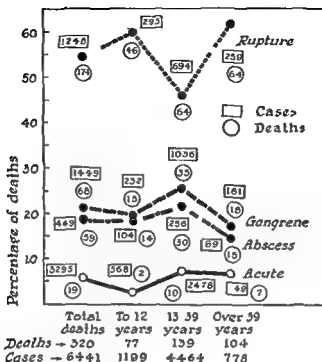


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pendicitis for complicated cases only since they compose the group in which the mortality is concentrated (fig 77) There certainly seems no justification for the occasional practice of eliminating from the mortality such causes of death as embolism pneumonia diabetic coma and anaesthesia and every justification for rigid adherence to the rule that a death following acute appendicitis must be classified as a death due to that disease

It must also be borne in mind that the true mortality of acute appendicitis is not the mortality of experienced and competent surgeons whose death rates even in complicated and advanced cases are often amazingly low The true mortality is not even the mortality of average surgeons It is rather the average mortality of all surgeons and to it should be added though it seldom is the average mortality of all physicians The term hospital mortality as many writers have pointed out is a misnomer Most of the deaths from acute appendicitis originate outside of the hospital and the surgeon though he bears the greater responsibility has least to do with them since the mortality is determined above everything else by the proportion of patients admitted with appendices already ruptured (fig 71 p 337)

### THE ERROR IN MEDICAL LITERATURE

In 1932 when Boland made his comprehensive study of acute appendicitis in Atlanta he called attention to the fact that only five papers on that subject had been read before the Section on Surgery of the American Medical Association during the preceding 15 years the last of them 5 years before Furthermore his review of the literature showed on the basis of the titles listed in *The Quarterly Cumulative Index Medicus* that only a third of the articles on appendicitis published between 1916 and 1930 concerned the acute disease

In the 15 years since Boland's study was made a great deal of attention has been paid to acute appendicitis and numerous articles have been written on it There is a rather general tendency however to emphasize its complications rather than the disease itself A special issue of a national surgical journal for instance was recently devoted to the general subject of diagnosis in surgical states The articles on carcinoma of the stomach empyema goiter and other diseases dealt with the diagnosis of the primary condition Not so with the article on acute appendicitis It was a valuable contribution to the diagnosis of the complications of acute appendicitis chiefly peritonitis but a young medical student who examined that journal would have an extremely warped

view of the primary disease. It is refreshing to find recent articles by such distinguished surgeons as Stone and Elman emphasizing acute appendicitis as such and putting its complications into their proper place.

Part of the emphasis on the complications of acute appendicitis is undoubtedly due to the teachings of Bower who has probably done more than any living person to demonstrate the means by which the mortality of the disease can be reduced. One hesitates therefore to take issue with him but it is difficult to agree with his ideas (1) that medical men should cease to write about acute appendicitis (2) that medical editors should cease to accept articles dealing with it and (3) that all the attention should be concentrated instead on rupture and peritonitis. In the sense that peritonitis is the cause of most deaths in acute appendicitis his concept is correct but that situation does not prevail because too little attention is paid to peritonitis. It prevails because too little attention is paid to acute appendicitis while it is still acute appendicitis. Appendiceal peritonitis develops because physicians fail to recognize the syndrome of the uncomplicated disease the spreading of the gospel of which has been Bower's chief contribution to the subject. From the standpoint of the literature McKenna's point of view is correct. He wrote: "Most of the teaching and writing is occupied with the management and treatment of the pathology of a disease that should never occur. In other words the best way to attack the disease is by the diagnosis and treatment of the early case rather than by the most skillful management of the late case."

Reginald Fitz also correctly pointed out the lack of attention which the disease receives among physicians. His father's original paper he noted had been read before the American College of Physicians and discussed by a group of distinguished internists including William Pepper and E. G. Janeway. In the next half century however only six papers on acute appendicitis had been read before the College and the surgeon had been left in complete charge of the disease which is obviously illogical since it is the physician and not the surgeon who usually sees the patient first. One has only to recollect how infrequent are articles on acute appendicitis in journals of internal medicine to realize the soundness of Fitz's charges.

Attention has frequently been called to the harm which careless statements in the literature may achieve if they are taken literally. Thus in one communication in an outstanding journal it was stated that acute appendicitis which has not reached the stage of perforation—a distinction not always as clear as the author implies that it is—is not as imperative and urgent as hemorrhage, strangulated hernia or perforated ulcer.

It is astonishing to find modern surgeons stating that if a patient is admitted after midnight operation should be deferred until morning in the absence of signs of spreading peritonitis—a diagnosis which one of them had stated categorically could not be made with any degree of safety. The rationale of this plan is that a fatigued operating team does not function well and that both team and patient are improved by the delay. The benefits to the patient inherent in this plan seem at least questionable and even more questionable seems the advice to administer morphine in the interim to rest the intestines, a policy which seems quite as likely to mask the advance of symptoms.

There is no doubt that the mortality of acute appendicitis always rises whenever there has been a flood of articles in the literature advocating expectant treatment for appendicular peritonitis chiefly because this type of therapy is also used for uncomplicated appendicitis by physicians who have misunderstood its purposes. The amount of damage wrought by those who write thus is precisely in proportion to their eminence in the surgical world.

*Textbook Presentations.* As part of its initial campaign to lower the death rate of acute appendicitis in that community, a committee of the Philadelphia Medical Society undertook a survey of standard textbooks, monographs and systems to determine how accurately the disease was described in them. Their conclusions were that the presentations were neither accurate nor adequate. In 1936 in a similar survey undertaken for the Orleans Parish Medical Society I arrived at almost similar conclusions.

Of the twenty-two books that I investigated five were issued between 1893 and 1914, twelve between 1930 and 1936 and the remainder between 1915 and 1929. The earlier texts which had been intended merely as standards of comparison were found in at least one instance to be considerably more alive to the seriousness of acute appendicitis than were several of the later texts.

In several of the recent texts the symptoms of peritonitis rather than of appendicitis were stressed. Perforation and gangrene were presented as if they were initial pathologic changes. The impression was given or the statement was actually made that the diagnosis of acute appendicitis is easy. Very little attention was paid to the atypical character of the disease and the frequent lack of correlation between clinical picture and pathologic process. The risk of purgation was either not stressed or was completely ignored. There was no strong emphasis on the importance of prompt operation and usually no emphasis at all on the fact that at any moment any patient's condition may become desperately

grave. The advice was often given that surgical or nonsurgical therapy be initiated on the basis of time elapsed between the onset of symptoms and the medical consultation. A surprising number of texts opened the discussion of treatment with a description of nonsurgical therapy which is bad psychology if no more. Almost without exception chronic and acute appendicitis were discussed under the same general heading. Obstructive appendicitis was almost completely ignored. Few texts even mentioned the serious character of acute appendicitis in advanced age though most of them mentioned its risks in extreme youth. In only a few was the forthright statement made that early operation is justified even on suspicion in acute appendicitis and that it remains justified if it has been performed on reasonable grounds even if the appendix is not seriously diseased.

A textbook written in 1914 stated that acute appendicitis is frequent, serious, alarming, multiple in its manifestations and aspects, demanding heroic measures for its cure, as clearly a surgical disease as a broken leg, a disease that demands the services of a surgeon as soon as it is suspected, a disease in which medical treatment is relegated in the best practice to the limbo of contemptuous oblivion. Until modern texts emulate those forthright statements, the medical literature from which students must secure their knowledge of the disease cannot escape a share of the responsibility for the mortality of acute appendicitis.

Unfortunately, all modern texts do not emulate those forthright statements. Incredible as it might seem, a synopsis of operative surgery issued in its second edition in 1947 recommended as the best management for acute appendicitis packing with ice and keeping the patient quiet and at rest for a few days until the acute inflamed condition subsides. One commends the point of view of the reviewer who dismissed the text with the remark that in view of the author's thoughts on acute appendicitis the book was not worth reviewing for scientific purposes.

*Chemotherapy and Antibiotic Therapy.* Medical literature is much to blame for the popular as well as the professional idea that new therapeutic agents have solved the problem of acute appendicitis. It would be in the highest degree unfortunate if the impression were to become general that the problem of the disease had been in any way solved by these methods. In some cases the results have been spectacular, but a more sober evaluation of the sulfonamides—the evaluation of penicillin was usually more dispassionate—has made it clear that their use is merely adjuvant to surgery. The second report of the Pennsylvania State Commission on Acute Appendicitis actually attributes to these agents a

share in a recent rise in the mortality of peritonitis following ruptured acute appendicitis.<sup>1</sup> Certainly these agents have not altered a single one of the fundamental problems of acute appendicitis in the correct concept of which peritonitis is classified as a regrettable and entirely avoidable complication and not as an integral part of the pathologic process.

### THE RESPONSIBILITY OF THE MEDICAL PROFESSION

Many physicians have felt their personal responsibility for teaching and preaching the facts of acute appendicitis. The vigorous writings of Ochsner, Deaver, Moynihan and Murphy still reward reading. Bailey recalled that John B. Murphy had taken it on himself to spend a part of every working day denouncing delay in the diagnosis and treatment of appendicitis as a pertinent factor in the high mortality. Some modern physicians are still assuming that burden.

It is not right or well the late Mont Reid wrote a few years ago for the profession to wait for enlightened public opinion to correct a situation the responsibility for which physicians should be courageous enough to assume though they might be warned as he personally had been that he was transgressing medical ethics in placing the onus of delay on physicians who referred cases to him. The profession Reid continued has no right for the sake of ethics to continue to protect by their silence physicians who are responsible for deaths from appendicitis. There is no longer any argument about how the disease should be treated and physicians who do not follow those rules should be censured by organized medicine exactly as a locomotive engineer who drove his train on the wrong track would be censured. Until that is done Reid concluded the public will continue to pay the price of an unnecessarily high death rate while professional ethics effectively defend the offender in our own profession. Murphy in 1914 made the same point when he wrote that the physician had no right to hold an opinion about appendicitis at radical variance with established facts.

The division of the profession into physicians and surgeons and the resulting division of disease into medical and surgical categories are partly responsible for the unhappy results in acute appendicitis as Fitz pointed out in 1939. To correct the situation he proposed three lines of attack. (1) Acute appendicitis should be taught in medical as well as in surgical courses. (2) A constant interest in the disease should be maintained in local medical societies. (3) The public should be instructed on the subject since the public is always willing to listen to sensible medical advice.

Anyone who does not believe that that sort of special teaching is necessary would do well to recall Elman's account of the interne  $\pi$  graduate of an excellent medical school who made a diagnosis of acute appendicitis in a child and advised observation. At the end of 24 hours in the course of routine ward rounds Elman saw the patient and immediately removed a gangrenous appendix. The Charity Hospital records bear impressive witness to the value of special emphasis on the disease to medical students. For several years the interne and resident groups contained a number of students from a school in which at that time  $\pi$  brief course in the preparation of medical papers was taught by a lay editorial assistant who based her teaching on a discussion of acute appendicitis in all its aspects. Again and again internes and residents who had taken this course would list acute appendicitis as a possibility in atypical cases and would sometimes hold to their opinion against the opinion of older members of the visiting staff. Several patients in the Charity Hospital series as a matter of fact owe their lives to the insistence of these young physicians.

Physicians need to be taught the value of positive action in acute appendicitis. They also need to be reminded of the disaster which failing to bear the disease in mind may accomplish. Bower and Clark in their first study of acute appendicitis at the Samaritan Hospital in Philadelphia found that the diagnosis had been made by physicians outside of the hospital in less than 3 per cent of the perforated cases with peritonitis although they constituted 45 per cent of the total number of cases. In the 6441 surgical cases studied from the New Orleans Charity Hospital 157 patients were seen outside of the hospital by physicians who delayed recommending surgery. 17 died. In 24 of the 158 fatal nonsurgical cases the same situation prevailed.\* In 70 of the 1852 surgical cases in which a purgative was taken purgation was recommended by a physician 15 patients died. The same situation prevailed in 11 of the 72 fatal nonsurgical cases in which a purgative was taken. Sometimes the physician said the patient did not have acute appendicitis. Sometimes the diagnosis was made but operation and hospitalization were not recommended. Sometimes no diagnosis was made. In 1 case fortunately unique the physician made the diagnosis of acute

This unfortunate situation continues. Stafford and Scott [Stafford Edward E. and Scott H. William "The mortality of appendiceal perforation" *South M J* 41:834-7 (Sept.) 1949] in a review of acute appendicitis at Johns Hopkins Hospital note that 12 of 23 patients who died of perforation of the appendix in the period 1939-47 had been seen by physicians more than 24 hours before they entered the hospital. The lay public they point out needs a better and a continuing program of education but equally obviously the physician needs to improve his index of suspicion.



appendicitis and ordered daily purgatives for a week. When one considers the possibility of error inherent in acute appendicitis even in the hands of well trained physicians who bear it constantly in mind the necessity of special education for less competent and less conscientious physicians needs no elaboration.

### Therapeutic Failure

Therapeutic failure is the ultimate cause of all deaths in acute appendicitis but that general statement does not cover the situation. Delay in instituting surgical therapy rather than failure to institute it and rather than the institution of incorrect therapy is the chief reason for the mortality of the disease. For that delay the patient is more often responsible than is the physician.

With the proviso that surgery be instituted promptly many physicians will be in complete agreement with Grey Turner's remark that details of technique do not alter the outcome in acute appendicitis. At Charity Hospital as has been mentioned elsewhere (p 363) incisions other than the McBurney were most often reserved for cases in which diagnostic confusion had arisen or complications had already developed and the statistics (fig 75) must be interpreted in the light of those facts. Collected series show equally good results with the McBurney and with other incisions with and without inversion of the stump and with various techniques of inversion of the stump. It is quite possible in fact that too much emphasis on what are after all unimportant details has sometimes obscured the three most important considerations in acute appendicitis: (1) how soon the patient is seen after the onset of his illness, (2) whether or not he has taken a purgative and (3) how promptly operation is done.

At Charity Hospital as elsewhere the mortality for simple appendectomy was lower than that for appendectomy and drainage or for incision and drainage and the mortality for incision and drainage was lower than that for cecostomy or enterostomy (fig 74 p 362). The latter procedure as already pointed out (p 301) is no longer necessary because constant intestinal decompression accomplishes the objective more efficiently. The mortality attached to most surgical procedures however is more often an index of the severity of the pathologic process than a real indication of their value. The mortality of primary colpotomy and proctotomy in the Charity Hospital series (11 of 26 cases) does however fairly raise the question as to whether this method of treat

ment is as valuable as some observers suggest that it is. It also raises the question as to whether the formation of a cul de sac abscess is a really desirable outcome of rupture of the appendix and generalized peritonitis treated expectantly.

The arguments for and against conservative therapy in appendicular peritonitis have been fully discussed (p. 203). In the hands of careful practitioners it has undoubtedly aided in the reduction of the mortality in this condition though it has brought it no lower than the mortality associated with prompt surgery. It seems fair to say however that under certain circumstances expectant treatment is definitely contra-indicated. It is employed on doubtful or actually dangerous indications when it is used in young children, in individuals advanced in years, in pregnant women, in patients who have taken purgatives, in very toxic patients, and in patients in whom localization is not clearly occurring. It is certainly of dubious wisdom when it is employed within the first 24 hours of illness, or even within the first 48 hours, though it is not safe generally speaking to institute therapy in relation to the duration of illness.

Part of the mortality of acute appendicitis can probably be explained by the practice of operating on patients in bad condition without proper rehabilitation. A delay of a few hours in such cases, in spite of the urgency of the disease, is often lifesaving. In a small proportion of cases death can be explained by failure to carry out proper postoperative therapy.

It is doubtful, granting the premise that anesthesia is competently given, that any one type is superior to any other type. The figures from the New Orleans Charity Hospital are not suitable for comparative purposes (fig. 78). Cyclopropane or some other inhalation anesthesia is used routinely on a few services in that institution, but otherwise this type of anesthesia is reserved for patients who are poor risks, and there is a general preference for spinal analgesia among residents and other surgeons who handle most of the emergency surgery. Nonetheless the mortality for spinal analgesia and the mortality for inhalation anesthesia were substantially the same, 4.66 and 4.67 per cent, respectively. Local analgesia was attended by the prohibitive mortality of 50 per cent, which merely means that its use was confined to patients whose condition was regarded as desperate.

It is generally true that the mortality of a skilled and experienced surgeon is lower than that of the inexperienced surgeon or of the unskilled and inexperienced occasional operator, and that his morbidity after operation is lower. There is also no doubt, as Collins points out

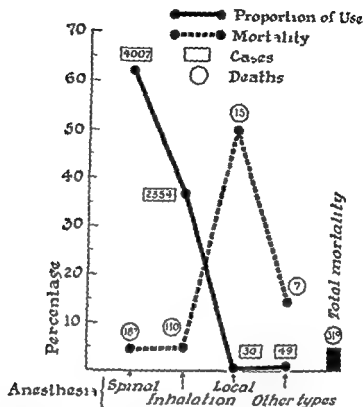


Fig. 78 Proportion of use of various types of anesthesia with related distribution of mortality in 6441 surgical cases of acute appendicitis

that thinks to the indifference of the usual hospital staff internes and residents is related in the fallacy that anybody can remove an appendix. Part of the belief is probably psychologic and is due to the habit of letting young men in training begin their supervised and independent operating on patients with appendicitis which is usually of the so called chronic variety without duly instructing them in the dangerous aspects that such cases may assume.

There is no doubt in short that criminally bad surgery is responsible for a portion of the mortality of acute appendicitis, but also no doubt that the proportion is fractional. The charge occasionally made that young surgeons are largely responsible for the continuing high death rate of the disease is simply not true. A poor surgeon who operates early is always better for the patient than a skilled technician who operates late and a young surgeon who loses no time in refinements of diagnosis often compensates for his lack of experience by his appreciation of the urgency of the disease.

## MORTALITY IN SPECIAL GROUPS

The good results possible in acute appendicitis under optimum conditions of treatment are seen in reports from such institutions as student infirmaries Army and Navy hospitals and closed cities such as Oak Ridge in which according to Hays report there were no deaths from acute appendicitis over a 2 year period in a population that varied from 25 000 to 75 000 It was rather surprising however to find that of the 357 appendectomies handled by the centralized medical personnel at Oak Ridge 78 of which were for subacute disease there were 12 instances of perforation with generalized and 11 instances of perforation with localized peritonitis and that in 177 other cases the appendix was either suppurative or gangrenous Since physicians were constantly on call the high proportion of complicated cases may probably be interpreted as indicative of the speed of progression of the pathologic process in this disease

That good results are possible even under less propitious circumstances is evident from the decreasing mortality of acute appendicitis in Philadelphia (p 419) which is directly attributable to the campaign of public health instituted by Bower and from the improved results in Cleveland reported by Green and Watkins Jennings and his associates who reported a mortality of 1.9 per cent in 1 680 consecutive cases of acute appendicitis from the Beth El Hospital in Brooklyn pointed out that almost 58 per cent of their patients had been admitted within 24 hours of the onset of illness The reason for this unusual showing is clear Although the hospital population consists chiefly of lower middle class or actually poor patients many of whom were on relief during the period covered by the survey all had been subjected to a high degree of education by various social and medical groups concerning the possible risks of abdominal pain

Even when medical care is readily available on the other hand and even in a population group that from the medical standpoint should be the most intelligent of all results are still far from perfect Fitz, commenting on 142 appendectomies performed among 2 840 internes and residents who had graduated from medical school in 1937 assumed probably correctly that most of them were prophylactic or for recurrent disease apparently all were not for there were 3 deaths Every year several physicians are listed in the annual classified list of deaths as dying from appendicitis

*Age and Race* The mortality of acute appendicitis according to age

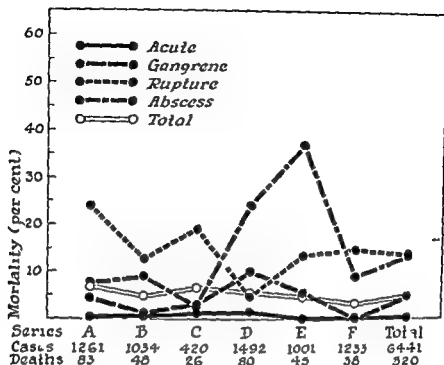


Fig 79 Serial mortality in relation to pathologic process in 6441 surgical cases of acute appendicitis

groups has been discussed in such detail under the appropriate headings (pp 335 and 353 figs 71 72 pp 337 340) that it is necessary here only to report that the disease carries an unduly high mortality in young children and an inordinately high mortality in middle and late life. Whether the higher rates at these periods of life are due to inherently more serious disease to diagnostic difficulties to delays in treatment introduced by the higher proportion of atypical cases or to other causes the end result is the same. It is important to note that although the decrease in mortality apparent in successive recent studies from the New Orleans Charity Hospital (fig 79) are shared by all groups the disproportion in the mortalities of the various age groups still continues (fig 80).

Numerically deaths from acute appendicitis are fewer among Negroes than among white persons for two reasons (1) that the Negro population of the country represents only 10 to 12 per cent of the total population and (2) that acute appendicitis is apparently less frequent in Negroes than in white subjects. A most misleading impression however is created by the use frequently made of these figures.

All reported series show that the mortality for acute appendicitis is

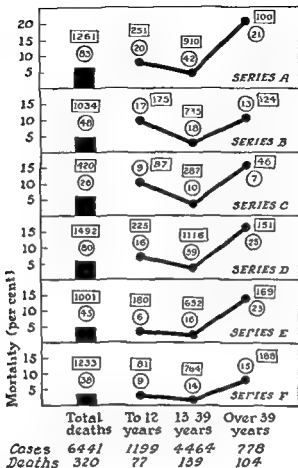


Fig. 80 Serial mortality in relation to age in 6441 surgical cases of acute appendicitis

much higher in Negroes than in white subjects. At the New Orleans Charity Hospital Negroes furnished over the almost 16 year period studied less than 30 per cent (28.8 per cent) of the cases but more than 40 per cent (42.4 per cent) of the deaths. Incidentally the proportion of white to Negro admissions at Charity Hospital which formerly averaged 55:45 has recently been reversed and is now approximately 45:55. In the last series of cases studied covering the period 1941-1945 Negro and white cases have come much closer together 578 to 655 respectively which means that in this period the Negro incidence of the disease was 46.9 per cent. The Negro mortality however (25 cases against 13 white cases) represents 66 per cent of the total mortality, the actual mortality being 4.32 per cent for Negroes against 1.98 per cent

for white patients. Regardless of such explanations as longer delay in seeking medical aid and a higher incidence of purgation and greater ignorance among Negro physicians (a lamentable but demonstrable fact directly attributable to inadequate facilities for training Negro physicians) there seems some reason to believe that acute appendicitis is an inherently more serious disease in the Negro than in the white subject. If the assumption is correct the corollary is also correct that there is even more need for haste in the diagnosis and treatment of acute appendicitis in the colored than in the white race.

*Previous Attacks* Any death that occurs in a patient who has had a previous attack of acute appendicitis and who has not been warned or who has not heeded the advice to have his appendix removed is an unnecessary death. Even more unnecessary are the deaths that occur in patients with rupture of the appendix who have been treated conservatively or who have been treated only by drainage and who have failed to return for removal of the appendix. In fact the difficulty of making the patient return for appendectomy is admitted even by those who advocate the method to be one of the objections to conservative therapy (p. 209). For some reason the mortality of acute appendicitis is less high among patients who have had previous attacks (fig. 73 p. 342). As has been pointed out, acute appendicitis may be expected to become less frequent and the number of fatal cases to become numerically less as more and more persons reach adult and late life with the appendix already removed because of the increasing incidence of interval appendectomy.

*Duration of Illness, Purgation and Pathologic Process* The mortality of acute appendicitis in the last analysis is related to the stage of the pathologic process. The pathologic process excluding the small proportion of cases in which the disease is of such violence that nothing could save the patient is related to two other factors, namely the duration of illness (symptoms) and whether or not a purgative has been taken.

The mortality of acute appendicitis rises progressively as the duration of the illness in terms of clinical symptoms increases (fig. 72 p. 340). Some individuals by what Heid calls the Grace of God may exhibit less serious pathologic changes at the end of a week than others exhibit at the end of a day. A patient with bacterial appendicitis is likely to exhibit less serious changes hour for hour than one with obstructive appendicitis (p. 135). Pathologic changes are always in progress before there is any clinical manifestation of their appearance. The process is unusually rapid in children, individuals in middle and late life, Negroes

and pregnant women. Making allowance for all of these considerations however the hour and on the clock determines the stage of the pathologic process when the patient is first seen and therefore determines the mortality.

All studies bear this out. Reid for instance observed in 1938 that in Cincinnati the average lapsed time before medical consultation was 91.2 hours while in Philadelphia it was 49 hours. It was easy to explain therefore why the death rate in Cincinnati was then 30 per 100,000 against 11 per 100,000 in Philadelphia.

The figures reported by Bower in 1931 are still generally true. When operation was performed within 24 hours 1 patient in every 24 died. When it was performed within 48 hours 1 in every 17 died. When it was performed within 72 hours 1 in every 13 died. When it was performed after that time 1 in every 9 died. In the hospitals of New Orleans in 1935 1 in every 39 persons with acute appendicitis died when operation was performed within 24 hours 1 in every 33 when it was performed within 48 hours and 1 in every 8 when it was performed after that time. In terms of pathologic process these chronologies mean that the percentage of patients admitted with perforation of the appendix determines the final mortality of acute appendicitis.

Reginald Fitz in his first paper on acute appendicitis in 1886 pointed out that a cathartic or laxative may be the means of at once eliciting a general peritonitis and that statement still holds though there is an occasional tendency observed in the literature to belittle the deleterious effects of purgation in this disease. Whether purgation increases the percentage of ruptured cases or hastens the process of perforation or prevents localization or merely delays diagnosis and treatment it would seem that there can be no argument about its evil effects.

Among experimental studies of purgation in acute appendicitis is a particularly convincing one by Bower who used over 700 dogs. In one group of animals the appendix and mesentery were tied off 50 per cent recovered following abscess formation. In a second group of animals subjected to the same procedure and given an ounce of castor oil 24 hours later 68 per cent died. In a third group in which the same medication was given immediately after operation 78 per cent died. In a fourth group in which the castor oil given immediately after operation was increased to 2 ounces 91.67 per cent died. Although the pathologic process produced by ligation of the appendix and mesentery of the dog is not precisely analogous to clinical appendicitis it would seem that



experiments such as these supply any proof which might be needed of the risks of lavative induced or therapeutic peritonitis

Clinical proof is also extremely convincing though histories are notoriously inaccurate on this point At the New Orleans Charity Hospital the mortality in surgical cases in which purgatives had been taken was 7.5 as compared with 3.9 per cent in the remaining cases in many of which it can be assumed that purgatives had also been taken the facts simply not being recorded When the purgative was repeated as it was from 1 to 14 times in 351 cases there were 52 deaths 14.81 per cent Purgatives were also stated to have been taken in 72 of the 158 fatal nonsurgical cases in the Charity Hospital series and were repeated from 1 to 5 times in 44 of these

In a study made in 1936 from all the New Orleans hospitals 1 in every 21 patients who apparently did not take a purgative survived operation against 1 in every 11 when one purgative was taken and 1 in every 7 when purgation was repeated<sup>1</sup> There is no need to multiply these figures for they are evident in every reported series One interesting fact however emerges from Kelly and Watkins series in which 67 per cent of the patients who died from the immediate causes of acute appendicitis were known to have taken purgatives The tendency of self medication apparently is the result of economic conditions during the depression rose from 24 per cent before 1930 to 42 per cent after that year It seems reasonable to assume that part of the recent improvement in the mortality of the disease may be attributable to improved economic conditions which permit prompter medical attention The crowded conditions in private hospitals all over the country suggest the validity of the assumption The physician's part in the mortality of acute appendicitis due to purgation has been discussed elsewhere (p. 405)

No blame can be attached to the physician who does not always make the diagnosis of acute appendicitis but he is assuredly well deserving of censure if he advises purgation for abdominal pain of the origin of which he is not perfectly positive If a purgative were never given until that certainty had been attained and if enemas were also withheld the mortality of acute appendicitis would undoubtedly show a much further decrease

Generally speaking whether the patient lives or dies depends chiefly upon the pathologic process present and chiefly upon whether or not the wall of the appendix is intact when he is first seen (figs. 71, 77, 79 pp. 337, 399, 410) That statement, however, needs some qualifications

Gangrene in many instances played an important part in the mortality at the New Orleans Charity Hospital even when the appendix was intact and considerably increased the mortality when it was present in association with rupture of the appendix McClure and Altemeier among others have also emphasized the dangers of the gangrenous appendix Gangrene was present in 46.4 per cent of their fatal cases in 46 per cent of all cases of spreading peritonitis and in 68.7 per cent of the fatal cases of spreading peritonitis

It has been pointed out at length elsewhere (p. 206) that abscess formation is not the consummation so devoutly to be wished as some articles in the literature might suggest

It is perfectly proper to emphasize the curable nature of early acute appendicitis. It is not a preventable disease though it is the most readily curable of all serious surgical diseases. With that emphasis however should go considerably more emphasis on the other side of the picture. Acute appendicitis in its early stages is not a lethal disease but it is potentially lethal. The statement that in its early stages it is not a dangerous disease is not true at all. How long will the stage of curability last? In the particular case under discussion will the disease go on to resolution if operation is not done promptly or go on to perforation and peritonitis? These are questions which no physician however skilled he may be can answer except by the law of averages and the law of averages as Lord Moynihan once wrote is a poor peg on which to hang a man's life. As Murphy wrote in 1916

Let us return to our ideal early operation is the only safe practice. Distrust of the inflamed appendix is the only safe surgical frame of mind. By operation we take the course of the disease into our own hands. By not operating we leave it in the hands of a blind and often terribly cruel fate.

The special dangers of obstructive appendicitis have already been emphasized (p. 135) but it might be repeated here that in this type of disease the pathologic process amounts to intestinal obstruction that there is little or no tendency to spontaneous reversal as there sometimes is in bacterial appendicitis that symptoms are misleading because in spite of the frequent violence of the abdominal pain there is an absence of the usual constitutional reaction that gangrene and rupture occur with unusual rapidity and finally that because of the speed of the process and the lack of reaction in the serosal coat of the organ rupture is likely to occur into a totally unprotected peritoneal cavity.

## MORTALITY IN RELATION TO DIAGNOSTIC DIFFICULTIES

The diagnostic difficulties of acute appendicitis have been discussed in sufficient detail (p 137) to need no iteration beyond the statement that part of the mortality of the disease is unquestionably due to the light hearted belief that diagnosis is a simple matter. Actually no other disease in the whole field of medicine or surgery can present greater problems which have not been simplified by such dogmatic statements as Murphy's that fever is a cardinal symptom or as Moynihan's that pain is always the first symptom. Failure to realize that the disease can occur coincident with any other illness and under any conceivable circumstances is another prolific source of mortality which also has been emphasized sufficiently elsewhere (p 157).

This would seem a logical place to point out again that one should view with wholesome respect the crises of appendicitis diagnosed as acute which prove at operation to be subacute or not acute at all. Appendectomy in such a case is an unnecessary emergency. It is true. Perhaps in some instances the surgeon's zeal outruns his discretion. In many if not in most cases however the clinical syndrome is such that there is every justification for recommending immediate surgery and many times the patient may seem in distinctly worse condition than the patient with true acute disease or even the patient with complicated acute appendicitis.

The point is worth laboring. If we continue to teach and preach as we should that immediate operation is justified on the reasonable suspicion of acute appendicitis or even more important on the impossibility of excluding the diagnosis then we must expect a certain proportion of operations that in one sense are unnecessary. On the other hand to justify such operations one need only recall the really unnecessary mortality in acute appendicitis due to failure to operate because a precise diagnosis could not be made or because the patient's symptoms were so mild that it seemed impossible that they could arise from any serious disease.

Comparisons of early and recent statistics for acute appendicitis (fig 76) are inaccurate for many reasons. The assumption is warranted that part of the apparent increase in the appendicitis death rate in the first quarter of the century is due to the inclusion in the appendicitis figures of many cases formerly included in the group of deaths from unknown causes or from peritonitis deaths from unknown causes showed a steady decline during the period in which the death rate

from appendicitis was showing a steady increase Lovelind who charted the figures of the Bureau of Vital Statistics over a period of years pointed out that it took 17 years for the medical profession to adjust itself to Fitz's discoveries about acute appendicitis Appendicitis was first listed as a cause of death in the vital statistics in 1892 in which year peritonitis had its greatest recent prevalence but it was not until 1903 that the effect of its inclusion was first apparent In 1925 appendicitis reached its maximum prevalence and peritonitis its permanent minimum prevalence

#### MORTALITY IN RELATION TO MISCELLANEOUS CAUSES

Climate altitude and race and sex distribution have little or nothing to do with the appendicitis death rate The fact that for many years the highest death rate for appendicitis in the United States was in the Rocky Mountain states is more readily explained by the fact that the territory is sparsely settled and that hospitals are correspondingly far apart than by climatic or other conditions

The death rate for males is usually somewhat higher than for females but in the Charity Hospital series the respective mortalities were 4.93 and 5.04 per cent Males furnished 64.9 per cent of the incidence and 61.2 per cent of the mortality

When statistics for acute appendicitis are discussed from the standpoint of rural and urban communities an immediate source of error is apparent namely the concentration of cases and therefore of deaths in cities with hospital facilities This concentration usually makes the rate for the city unduly high and the rate for the adjacent country unduly low

For some reason still to be elucidated rates for comparable cities in the United States vary consistently The low rates in some cities however are easy to explain For many years the appendicitis death rate in Philadelphia has been consistently lower than in any other American city of comparable size The explanation is the highly practical one that since 1929 there has been a determined and consistent effort to reduce the mortality of the disease by education of both profession and public and that the cumulative effects of that effort become more apparent with each successive year

Modern endeavors to control the mortality of acute appendicitis by campaigns of public education derive from the work of Dr John O Bower of Philadelphia Many communities have made sporadic endeavors to carry out similar campaigns and some cities such as

Cincinnati, have undertaken sustained campaigns Philadelphia however remains the outstanding example of what can be done in this regard The campaign has now been extended to the whole state of Pennsylvania and two state wide reports have been issued by the Appendicitis Committee of the State Medical Society<sup>1 2</sup>

### COMMUNITY ENDEAVORS

*The Philadelphia Effort* The story of the Philadelphia effort is worth telling in some detail In 1927 when Dr Bower was a young surgeon in the Samaritan Hospital in Philadelphia certain facts about acute appendicitis began to impress him more and more He saw that the death rate had been constant in the disease over many years or that actual increases had occurred He saw that the greatest wastage of life was in young persons in the prime of life and of most value to the community He saw physicians and surgeons complacent about the death rate in the disease chiefly because they did not know what it really was He saw the best surgical skill fail to save patient after patient Finally he decided that something had to be done about it

The practical outcome of his reflections was an analysis of hospital records of appendicitis and in January 1927 he made his first report on 1010 cases before the Philadelphia College of Physicians He pointed out that 1 in every 12 persons with acute appendicitis in Philadelphia could expect to lose his life More important he was able to show why These people died of peritonitis not of appendicitis Still more important he could show what caused most cases of peritonitis The chief causes were delay in diagnosis and operation plus the taking of purgatives for abdominal pain Most important of all both of these causes were preventable

With the co operation of the Philadelphia College of Physicians the appendicitis records of 27 hospitals in that city were studied for 1928-9 They showed a mortality of 5.99 per cent They showed that 42.7 per cent of the patients had been admitted with generalized peritonitis that the duration of illness averaged 56.5 hours for all cases and 95.6 hours for the fatal cases that a large proportion of the patients who died had taken purgatives and that many of them had repeated them

In the face of these facts education of the public was obviously the clue to the reduction of the mortality and since 1929 the physicians of Philadelphia have carried on a continuous campaign of publicity to teach the citizens of that city the three most important considerations in acute appendicitis (1) Any abdominal pain may prove to

be due to acute appendicitis (2) A purgative or laxative should never be taken for abdominal pain (3) Early operation in acute appendicitis is lifesaving

Within a year after the campaign started the mortality of acute appendicitis in the 27 hospitals originally studied (plus 1 additional hospital) had been reduced from 5.99 to 4.81 per cent. The proportion of patients entering with generalized peritonitis had been reduced from 42.7 to 35.45 per cent. The duration of symptoms before treatment had been reduced from 56.5 to 48.57 hours and in the fatal cases from 95.6 to 68.0 hours. In 1939 the mortality in the same 28 hospitals had fallen to 3.54 per cent. The death rate for acute appendicitis in the whole city and later in the state as the campaign was extended to it has fallen progressively since and today the citizens of the whole Commonwealth of Pennsylvania and particularly of the city of Philadelphia have a better chance of surviving acute appendicitis than the citizens of any other state and city in the country.

The campaign was initiated by the local medical profession of Philadelphia and is still conducted by physicians but druggists, schools, business houses, newspapers and the radio soon began to co-operate and finally the state medical society added its influence. The plan of the campaign has always been extremely simple and for the first five years the cost was only \$7,034, an average of \$1,500 per year distributed as follows: \$2,165 for 20,000 bulletins; \$400 for 20,000 public health facts; \$276 for 270,000 stickers; \$4,200 for salaries.

The aim has been to keep appendicitis constantly before the attention of both the profession and the public. Speakers before clubs, business houses, mothers' clubs and school children have emphasized the chief facts of acute appendicitis. That there is no way to prevent it but that deaths from it can be prevented if a physician is called for every abdominal pain which lasts 2 hours or more and if in the meantime the patient goes to bed, refrains from taking food, fluids or drugs of any kind and does not use icecaps, hot water bags or any other methods of self-treatment. The disastrous effects of purgation are clearly explained. After the talks, booklets are distributed to clubs and other organizations and each child in the school groups is given a sticker to be pasted in a book which he can carry home and show his family.

Posters are placed in stores, factories, public buildings and schools. Display cards are placed in street cars and buses. Public health facts are placed in many public places. Special articles are published in the newspapers and in high school and college journals. Addresses are made over the radio. Druggists co-operate by special window displays and

by placing on all purgatives and laxatives which leave their stores stickers furnished by the medical society that warn of the risks of purgation for abdominal pain. They also put leaflets into their packages. Life insurance companies distribute literature to local policyholders.

The Philadelphia Committee has correctly worked on the principle that campaigns of education are quite as necessary among supposedly educated and intelligent persons as among the ignorant and quite as necessary among financially well to do persons as among the poor and poverty stricken. A special attempt has been made to reach the high school and college groups in which acute appendicitis is most frequent. In 1935 Bower estimated that there were in the United States approximately 6 500 000 high school students of whom 58 000 were likely to develop acute appendicitis each year and of whom 2 906 were likely to die unless they could be told the true facts of the disease.

Bower has repeatedly stated that the reason the mortality for appendicular peritonitis is so much lower in Philadelphia than in most other communities is because the conservative method of treatment is generally employed for it. Without entering at this time upon that highly debatable subject it seems far simpler to explain the lowered mortality by the reduction in the duration of the illness before the patient is seen and by the greatly reduced incidence of purgation in appendicitis. Such a story as the following related by Smyth seems to prove the point.

A 5 year old child was brought into a Philadelphia hospital by the parents accompanied by a 10 year old sister who had steadfastly refused to obey her mother's instructions to go to the drug store for a purgative for the stomachache from which the younger child was suffering. Her reason was that the school physician a few days before had talked to them about the dangers of taking purgatives for abdominal pain. The older child the mother said contended that the younger one had appendicitis and kept yelling until we all came to the hospital. A fulminating acute appendix was removed within the hour.

*Other Community Endeavors.* Other cities have since followed the Philadelphia example always with a reduction of the appendicitis mortality. In 1936 for instance the annual Longer Life Week of the Orleans Parish Medical Society was devoted to the subject of acute appendicitis the campaign being based on the Philadelphia plan though necessarily compressed because of the brief duration of the effort. During 1936 the number of deaths from appendicitis in New Orleans numbered 95 as compared with 113 the previous year and the eminent statistician F. L. Hoffman<sup>4</sup> attributed the decrease probably correctly to that single week's campaign. The latest report by Green and Watkins

on appendicitis in Cleveland for a 12 year period shows a decrease in the crude mortality rate from  $5.67 \pm .62$  in 1933 to  $2.89 \pm .38$  in 1941. The writers attribute the improvement to less delay, more favorable condition of the appendix at operation and the changes in anesthetic and drainage. Of those factors undoubtedly the first two played the major role.

#### ACUTE APPENDICITIS AS A PUBLIC HEALTH PROBLEM

Public minded insurance companies and drug houses have done a great deal of good in the campaign of education concerning acute appendicitis the former by distributing special material to their policy holders and both groups by publishing material in national lay magazines depicting the cardinal facts of the disease and the disastrous effects of delay and purgation. That policy as was pointed out by D. B. Armstrong, Third Vice President of the Metropolitan Life Insurance Company, reduced the mortality from this disease among policyholders of the company by 58 per cent (from 13.2 to 5.5 per cent between 1933 and 1941) and reduced death claims due to this cause from \$3,032,000 to \$2,000,000 in spite of a great increase in the amount of insurance in force.

It is of course essential that publicity be correct and lay presentations are unfortunately not free from errors. A recent excellent advertisement for a drug company is marred by the inference that in the absence of fever no concern need be felt about the presence of abdominal pain so far as acute appendicitis is concerned. The fallacy of that generalization has been discussed elsewhere (p. 13). Discussions for the laity of the leukocytosis in acute appendicitis which is a highly variable factor (p. 132) of differential diagnosis and of the merits of various incisions are confusing and unnecessary. It would be much safer merely to inform the public that there is sometimes no way of determining that a disease is not acute appendicitis and that in such cases the patient's interests are best served by an operation even if it proves to be unnecessary.

An article in a lay journal by a prolific and rather sensational writer on medical subjects indicated that persons who kept fit, corrected focal infections and had an annual physical examination were less likely than others to develop acute appendicitis. The author stated that an enema would not hurt the patient regardless of the cause of his pain which is far from true and actually dangerous. Equally dangerous was the statement that if the pain did not disappear after the enema the diet should be restricted to fluids, actually it should be restricted to nothing at all.



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since any kind of intestinal activity may be disastrous. The final statement that appendectomy is so simple that any surgeon can perform it competently is very far from the truth. The mortality of the interval operation as a matter of fact is largely due to inept surgery though the statement might properly have been made that in acute appendicitis operation early by an inexperienced surgeon is better than operation late by a master.

Lay publications have also done a great deal of harm—and are continuing to do it—in respect to what can be achieved by the new therapeutic agents. A journal with it is said the largest circulation in the country published an article concerning according to the title the reduction of the mortality of appendicitis to zero. Actually it dealt with the use of sulfanilamide in appendicular peritonitis which it suggested had now lost all its terrors. There was no indication in this article that appendicitis has other complications than peritonitis, that all cases of peritonitis do not respond to sulfonamides, and that the sulfonamides are dangerous drugs. Actually, the intraperitoneal use of sulfanilamide with which the article was concerned is now largely discredited but aside from that fact it seems dubious wisdom to teach the public that these or the antibiotics are harmless miracle drugs. It would be most unfortunate if lay journals were to continue to spread this sort of half-baked information. If they do the curious situation may arise in which lifesaving measures may actually become one of the factors of mortality in acute appendicitis by letting an already ignorant public gain the impression that peritonitis itself is now completely controlled even though it may be more desirable as the author of the article just referred to does state in a sort of aside to have one's appendix removed before rupture.

The trend in the mortality of acute appendicitis is downward practically everywhere chiefly there seems little doubt because patients are being seen earlier and the proportion of simple acute cases of the disease is successively greater. On the other hand the battle is not won. At the New Orleans Charity Hospital while the proportion of complicated cases has steadily decreased in the almost 16 year period for which the figures have been collected (fig. 81) the decrease is not entirely constant the proportion of simple acute cases was less in the two latest studies (E and F) than in the preceding study (D). The fact that the mortality was successively lower in every series studied does not remove the disturbing implications of this fact. Continued vigilance is obviously essential to hold the gains which have been made.

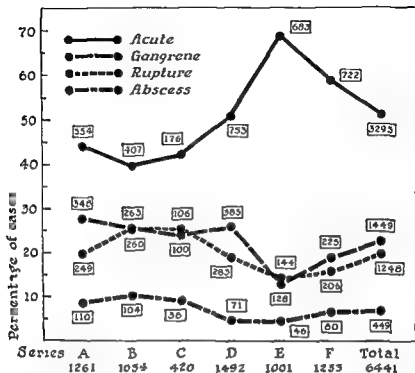


Fig 81 Proportionate distribution of the pathologic process in six serial studies of acute appendicitis totaling 6 441 surgical cases

Frederick L Hoffman some years ago made the unqualified statement that appendicitis is a public health problem of major importance

The time seems to have come for a national organization for appendicitis prevention corresponding to the tuberculosis movement and the cancer movement to visualize the facts of the situation for the instruction of the laity as well as of the medical profession emphasizing on the one hand the supreme importance of the earliest possible surgical treatment in acute cases and on the other the danger of taking purgatives with or without medical advice

Bower states that if a publicity campaign of increasing intensity can be waged against purgatives and delayed hospitalization in acute appendicitis within a decade spreading peritonitis should be as rare in Philadelphia as typhoid fever is now and there seems no reason why that ideal should not be achieved in the whole country

The conclusion of the whole matter is found in the foreword of the first state wide survey of the mortality of acute appendicitis in Pennsylvania which was published in 1940 with Dr Bower appropriately as

chairman of the committee and which contains the analysis of 20 000 cases studied from records of hospitals throughout the state in 1937.<sup>1</sup> The report and the foreword which is entitled "The Problem Restated" comprises

a set of facts which force the thinking physician and surgeon to discard the old idea of appendicitis mortality if they have not already done so. There is no appendicitis mortality. The facts point out exactly where to find the major causes of death misquarling as appendicitis. After due allowance is made for unavoidable catastrophes the problem is *To Prevent Deaths from Spreading Peritonitis*. The Medical Society of the State of Pennsylvania in this report turns over to the physicians who first see the appendicitis patient and to the surgeons the results of the survey of these 20 000 cases which virtually constitute a road map with the death traps clearly marked. With the objective so plainly outlined it remains for the Commission to continue not only its Prophylactic Campaign remembering it is educating a people with new contingents constantly coming up but also to spread throughout our State Society membership the gospel of Better Management of Spreading Peritonitis for both Physician and Surgeon to become evangelists of Better Management of the Perforated Appendix or Spreading Peritonitis.

To that need be added only the statement that the best management of both the perforated appendix and spreading peritonitis is to prevent their occurrence by the proper management of early acute appendicitis that is by early operation and by abstinence from purgation which implies in turn continued education of the public in regard to these points.

<sup>1</sup> Report of the second state wide survey of acute appendicitis (23 000 cases studied from state wide hospital records of 1942) Conducted by the Medical Society of the State of Pennsylvania under the direction of the Commission on Acute Appendicitis Mortality John O Bower Chairman Pennsylvania M J 48 911-36 (June) 1945

<sup>2</sup> The mortality of acute appendicitis in the hospitals of New Orleans a report New Orleans M & S J 89 170-74 (October) 1936

<sup>3</sup> Report of the first state wide survey of acute appendicitis (20 000 cases studied from state wide hospital records of 1937) Conducted by the Medical Society of the State of Pennsylvania under the direction of the Commission on Acute Appendicitis Mortality John O Bower Chairman Pennsylvania M J 43 1143-74 (May) 1940

<sup>4</sup> Hoffman F L The appendicitis record of 1936 Spectator Oct 28 1937 pp 69-80

## References

- Abbott W O and Johnston C G Intubation studies of the human small intestine X A non surgical method of treating localizing and diagnosing the nature of obstructive lesions *Surg Gynec & Obst* 66 691 7 (Apr) 1938
- Abt I A Appendicitis in infants *Arch Pediat* 34 641 57 (Sept) 1917
- Aird Ian Acute non specific mesenteric lymphadenitis *Brit M J* 2 690 82 (17 Nov) 1945
- Allen Arthur W Interruption of the deep veins of the lower extremities in the prevention and treatment of thrombosis and embolism *Surg Gynec & Obst* 84 519 27 (15 Apr) 1947
- Allen Arthur W Linton Robert R and Donaldson Gordon A Venous thrombosis and pulmonary embolism *J A M A* 133 1268 76 (26 April) 1947
- Allen Philip D Acute appendicitis in children *J A M A* 109 121 5 (10 July) 1937
- Altmeier W A The bacterial flora of acute perforated appendicitis with peritonitis A bacteriologic study based upon one hundred cases *Ann Surg* 107 517 28 (Apr) 1938
- Altmeier W A and Holzer C E Primary torsion of the omentum *Surgery* 20 810 19 (Dec) 1946
- Altmeier W A and Wadsworth C L Penicillin—its use in surgery and influence on earlier types of chemotherapy *Surg Gynec & Obst* 84 540 52 (15 Apr) 1947
- Althausen T L Dextrose therapy in diseases of the liver *J A M A* 100 1163 7 (15 Apr) 1933
- Althausen T L Effects of the administration of glucose and insulin on the glycogen content of normal and experimental damaged livers *Ann Int Med* 6 193 200 (Aug) 1932
- Altschuler Emil A diagnostic sign for retrocaecal appendicitis *Lancet* 1 891 2 ✓ (16 Apr) 1938
- Altarex W C When should one operate for "chronic appendicitis? A study of 385 cases *J A M A* 114 1301 6 (6 Apr) 1940
- Andrews Edmund Do gripe seeds cause appendicitis? *J A M A* 27 1196-7 (5 Dec) 1896
- Arkush A S and Kosky A A 'The accuracy of diagnosis of appendicitis' *J Lab & Clin Med* 25 1276-87 (Sept) 1940
- Armstrong D B The probable influence of health education on the improving appendicitis mortality rate *Brit M J* 1 277 9 (6 Mar) 1943
- Arnheim E E and Neuhof H A lowered mortality in acute appendicitis and the basis therefor *Surg Gynec & Obst* 59 189 93 (Aug) 1934
- Aschoff Ludwig *Appendicitis Its Aetiology and Pathology* translated by Pether G C Constable & Company Ltd London 1932
- Ashburn L L Appendiceal oxyuriasis its incidence and relationship to appendicitis *Am J Pathol* 17 841 56 (Nov) 1941
- Aud Guy Deaths from acute appendicitis in Louisville *South M J* 34 914 17 (Sept) 1941
- Babbage E Deun McLaughlin C W and From R L Strain of right rectus muscle simulating acute appendicitis *War Med* 5 290 82 (Mar) 1944

- Babler E A Perforative appendicitis complicating pregnancy With report of a successful case *J A M A* 51 1310-14 (17 Oct) 1908
- Bier J L Rens R A and Arcus H A Appendicitis in pregnancy with changes in position and axis of the normal appendix in pregnancy *J A M A* 98 1359-64 (16 Apr) 1932
- ✓ Boley F W Acute appendicitis—a brief criticism *Ann Surg* 96 530-36 (Oct) 1932
- Boley Hamilton Diseases of the vermiform appendix in Mungot Rodney *Postgraduate Surgery* B Appleton Century Company Inc New York and London 1936
- Bain C G and Eagles H Peritonitis treatment with peptone broth *West J Surg* 49 449-51 (Aug) 1941
- Barber A H Acute mania from appendicitis *Brit M J* 1 162-3 (29 Jan) 1939
- Birlow Richard A Recovery from suppurative pyelophlebitis (correspondence) *Lancet* 2 134 (27 Jul) 1940
- Barnes Roger W and Hill Malcolm R Intestinal fistula *J A M A* 133 456-8 (15 Feb) 1947
- Baronofsky Ivan D Treloar Alvin E and Wingensteen Owen H Blood loss in operations a statistical comparison of losses as determined by the gravimetric and colorimetric methods *Surgery* 20 761-9 (Dec) 1946
- \* Bartels C D and Marcus Hansen E Serum therapy of appendicitis *Acta chir Scandinav* 92 1 16 (10 Mar) 1945 Abstract *J A M A* 129 1293 (29 Dec) 1945
- Bartlett M K Jones C M and Ryan A E Vitamin C and wound healing I Experimental wounds in guinea pigs II Ascorbic acid content and tensile strength of healing wounds in human beings *New England J Med* 226 469-81 (19 Mar) 1942
- Bartlett M K and Miller R H Acute appendicitis as a complication of carcinoma of the cecum *New England J Med* 222 783-4 (9 Mar) 1940
- Battle W H Modified incision for removal of vermiform appendix *Brit M J* 2 1360 (30 Nov) 1895
- Beardwood Joseph T Jr The abdominal symptomatology of diabetic acidosis *J A M A* 105 1168-72 (12 Oct) 1935
- Beck W C Koucky J D and Baker Morton Diagnosis and localization of intra abdominal abscesses by roentgenological methods *Am J Surg* 53 113-17 (Jan) 1942
- Behn R J Acute generalized suppurative peritonitis Treatment by intra abdominal lavage with ethyl alcohol (reduction of mortality from 50 to 4 per cent) *Am J Surg* 25 28-34 (July) 1934
- Berger Louis and Achs Samuel Perforation of the small intestine by the Miller Abbott tube *Surgery* 22 648-56 (Oct) 1947
- ✓ Berkeley William and Watkins Hury C Chemotherapy in the management of acute appendicitis *U S Nav M Bull* 42 1 6 (Jan) 1944
- Bernard H and Jomay J L'appendicite du vieillard *Monde med Paris* 44 866-74 (1 15 Sept) 1934
- Bernstein Mitchell and Goldsmith Ralph Bradycardia in appendicitis report of a case *Ann Int Med* 10 1716-21 (May) 1937
- ✓ Bernstein Phineas Ovarian tumors and diagnosis of acute appendicitis *Arch Surg* 37 1004-7 (Dec) 1938
- Bickham Warren Stone *Operative Surgery* W B Saunders Company Philadelphia and London 1935 5 59-60
- Bigger Joseph W Synergic action of penicillin and sulphonamides *Lancet* 2 142-5 (29 July) 1944

Bigham Roy H Jr, Myson Robert H and Howard John E. Total intravenous alimentation: its technique and therapeutic indications. *South M J* 40: 238-46 (Mar) 1947

Bissell A H. Trauma as factor in acute appendicitis. *Arch Surg* 17: 672-5 (Oct) 1928

Block F H and Michael M A. Acute appendicitis in complete transposition of viscera. Report of case with symptoms referable to right side. Mechanism of pain in visceral disease. *Ann Surg* 107: 511-16 (Apr) 1933

Boland Frank K. Results in treatment of acute appendicitis: review of 4270 cases in Atlanta. *J A M A* 99: 443-8 (8 Aug) 1932

Boldt H J. The management of laparotomy patients and their modified after treatment. *New York M J* 85: 145-53 (26 Jan) 1907

Bondarenko N T. Acute appendicitis in persons past forty. *Soviet khir* 8: 266-73 1936. Abstract *J A M A* 109: 343-4 (23 Jan) 1937

Bower J O. Acute appendicitis: Survey of its incidence and care in Philadelphia. *J A M A* 96: 1461-5 (2 May) 1931

Bower J O. Cause and prevention of increasingly high mortality in the acute surgical abdomen. *Am J M Sc* 174: 225-31 (Aug) 1927

Bower J O. Clinical and surgical aspects of spreading peritonitis complicating acute perforative appendicitis. *Minnesota Med* 23: 755-67 (Nov) 1940

Bower J O. A clinical pathologic classification of acute appendicitis and peritonitis complicating perforative appendicitis. *Am J Surg* 45: 66-71 (July) 1939

Bower J O. The lucid interval and acute appendicitis. *Am J M Sc* 195: 529-33 (Apr) 1938

Bower J O. Mortality of acute appendicitis: report of progress made in campaign for its reduction in Philadelphia. *J A M A* 99: 1765-8 (19 Nov) 1932

Bower J O. Spreading peritonitis complicating acute perforative appendicitis: routine operations versus scientific management. *J A M A* 112: 11-17 (Jan) 1939

Bower J O, Burns J C and Mengle H A. The bacteriology of spreading peritonitis complicating acute perforative appendicitis: A clinical and experimental study. *Surgery* 3: 645-57 (May) 1939

Bower J O, Burns John C and Mengle H A. Induced spreading peritonitis complicating acute perforative appendicitis. *Surg Gynec & Obst* 66: 947-61 (June) 1938

Bower J O, Burns J C and Mengle H A. Laxative induced spreading peritonitis complicating acute perforative appendicitis: results obtained with use of Perfringens Clostridium welchii antitoxin and a partially maintained glucose metabolic balance. *Am J Surg* 45: 221-9 (Aug) 1939

Bower J O, Burns J C and Mengle H A. Spreading peritonitis complicating acute perforative appendicitis: Experimental studies. *Arch Surg* 37: 751-9 (Nov) 1938

Bower J O and Clark J H. Controllable factors affecting mortality of acute appendicitis: fallacies of present day teaching. *J A M A* 63: 844-6 (10 Sept) 1927

Bower J O and Clark J H. Plan for reduction of mortality in acute appendicitis. *Pennsylvania M J* 33: 393-6 (Mar) 1930

Bower J O, Mengle H A and Paxon N F. Demonstration of antitoxin for the toxin of Clostridium welchii in the blood serum of patients and dogs that have recovered from peritonitis complicating acute perforative appendicitis. *J Immunol* 34: 185-93 (Mar) 1938

Bower J O, Terzian L A and Pearce A E. Changes in the blood and the composition of the peritoneal exudate in induced spreading peritonitis. *Arch Surg* 44: 1091-1102 (June) 1942



- Bowers W F Appendicitis with especial reference to pathogenesis bacteriology and healing *Arch Surg* 39 362 122 (Sept) 1939
- Bowers W F and Richard Norman F Rectus muscle strain simulating acute appendicitis *Mil Surg* 92 645 8 (June) 1943
- Bowers W F and Shupe L Acute appendicitis sequela of typhoid inoculation *Mil Surg* 90 413 (Apr) 1942
- Boyce Frederick F *The Role of the Liver in Surgery* Charles C Thomas Springfield and Baltimore 1941
- Boyd William *Surgical Pathology* 4th ed W B Saunders Company Philadelphia and London 1939
- Brennemann Joseph The abdominal pain of throat infections *Am J Dis Child* 22 493 11 (Nov) 1921
- Brennemann Joseph The abdominal pain of throat infections in children and appendicitis *J A M A* 89 2183 6 (24 Dec) 1927
- Brentano C Influence of insulin on glycogen formation from dextrose in normal animals *Klin Wchnschr* 18 42 6 (14 Jan) 1939 Abstract *J A M A* 112 1301 (1 Apr) 1939
- Breslin F J Gas bacillus infection of the abdominal wall in appendicitis *Am J Surg* 49 501 2 (Sept) 1940
- Bruce G G Diagnosis and treatment of acute appendicitis in children A review of 467 consecutive cases *Lancet* 1 1247-51 (3 June) 1939
- Brunn Harold Acute pelvic appendicitis *Surg Gynec & Obst* 63 583 92 (Nov) 1936
- Bryant J D Removal of the vermiform appendix Letter from New York (Domestic Correspondence) *J A M A* 8 55 6 (8 Jan) 1897
- Burge R E Dennis C Varco R L and Wangenstein Owen H Histology of experimental appendiceal obstruction (rabbit ape and man) *Arch Path* 30 481 503 (Aug) 1940
- Bullowa J G M McCabe E J and Wisluk S M Acute appendicitis in the exanthems *Am J Dis Child* 53 1029-38 (Apr) 1937
- Bumm E Zur Klinik der Greisenappendicitis *Deutsche Ztschr f Chir* 234 794 9 1931
- Bunch George H Mucoid disease of the appendix *Ann Surg* 121 704 9 (May) 1945
- Bunch George H and Adcock D F Giant faceted calculus of the appendix *Ann Surg* 109 143 6 (Jan) 1939
- Burford G E Pulmonary complications following 1333 administrations of cyclopropane *J A M A* 110 1057 92 (2 Apr) 1939
- Burgess C M Traumatic appendicitis *J A M A* 111 699 700 (20 Aug) 1938
- Butler Stuyvesant Feeney Neil and Levine M A The patient with heart disease as a surgical risk Review of four hundred and fourteen cases *J A M A* 95 85 91 (12 July) 1930
- Butsch Winfield L and Harberson James C Acute virus infection with nerve root involvement simulating appendicitis *J A M A* 123 405 7 (16 Oct) 1943
- Cantor M O Kennedy O S and Reynolds R P Use and abuse of intestinal decompression tube A study based on 200 cases *Am J Surg* 73 437 49 (Apr) 1947
- Carlson H A and Wilder Lucretia The Schilling hemogram in appendicitis *Arch Surg* 30 325 35 (Feb) 1935
- Carshaw R B On the character significance and prognostic value of peritoneal exudates *Brit J Surg* 3 8-38 (July) 1915
- Carter M N Left subphrenic abscess *Ann Surg* 110 562 77 (Oct) 1939

- Chenoweth Arthur I Appendicel abscess *Surgery* 14 702 7 (Nov) 1913
- Christopher Frederick and Jennings W K Certain factors in the operative mortality of acute appendicitis *Am J Surg* 18 111 18 (Oct) 1932
- Clado Appendice cecul anatomie embryologique anatomie comparée histologie normale et pathologique *Compt rend Soc de biol* 44 133 72 1892
- Coller F A and Brinkman H Studies on the reaction of the peritoneum to trauma and infection *Ann Surg* 109 912 54 (June) 1939
- Coller F A Campbell Kenneth N Vaughn Herbert F Lob L Vivian and Moyer Carl A Postoperative salt intolerance *Ann Surg* 110 533 42 (Apr) 1911
- Coller F A Dick V H and Maddock W G Maintenance of normal water exchange with intravenous fluids *J A M A* 107 1522 7 (7 Nov) 1936
- Coller F A Lob L Vivian Vaughn H H Kaldor N B and Moyer C A Translocation of fluid produced by the intravenous administration of isotonic salt solutions in man postoperatively *Ann Surg* 122 603 77 (Oct) 1915
- Coller F A and Potter E B The treatment of peritonitis associated with appendicitis *J A M A* 103 1753 8 (8 Dec) 1934
- Collins D C Acute retrocecal appendicitis based on seven hundred and fifty one instances *Arch Surg* 36 729 43 (May) 1939
- Collins D C Diverticuli of the vermiform appendix A study based on thirty cases *Ann Surg* 104 1001 12 (Dec) 1936
- Collins D C Etiological factors in acute appendicitis based upon a study of 3400 cases *Surgery* 5 267 70 (Feb) 1939
- Collins D C Hematuria associated with acute appendicitis *Urol & Cutan Rev* 42 22 4 (Jan) 1938
- Collins D C Mechanism and significance of obliteration of the lumen of the vermiform appendix *Ann Surg* 104 199 211 (Aug) 1936
- Collins D C The treatment of complicated acute appendicitis *M Rec* 150 127 9 (18 Aug) 1939
- Colp Ralph Chills in acute appendicitis an analysis of 2941 cases of acute appendicitis treated in Mt Sinai Hospital of New York City *Ann Surg* 85 257 62 (Feb) 1927
- Cumroe B I Non surgical causes of acute abdominal pain *Ann Surg* 101 438 44 (Jan) 1935
- Connell J E A Trauma and appendicitis *Surgery* 7 47 60 (Jan) 1910
- Cope Zachary *Clinical Researches in Acute Abdominal Disease* 2nd ed Oxford University Press London and New York 1927
- Cope Zachary *The Early Diagnosis of the Acute Abdomen* 9th ed Oxford University Press London and New York 1916
- Cope Zachary The prevention and early diagnosis of the acute abdomen *Brit M J* 1 69 (5 Jan) 1929
- Cope Zachary *The Treatment of the Acute Abdomen Operative and Post Operative* 2nd ed Oxford University Press London and New York 1929
- Copland S M and Mingolatta C J The mimicry of acute appendicitis by malaria *Rev Gastroenterol* 1 183 91 (Sept) 1934
- Corcoran William J Prenatal rupture of the appendix *Am J Dis Child* 39 277 80 (Feb) 1930
- Cosgrove E A Surgical complications of pregnancy *Am J Obst & Gynec* 31 469 79 (Sept) 1937
- Crisle C Jr Peritonitis of appendiceal origin treated with massive doses of penicillin Report of 50 cases *Surg Gynec & Obst* 83 150 62 (Aug) 1916
- Crocker W J and Valentine F H Hemography in the diagnosis of appendicitis based on 500 cases *J Lab & Clin Med* 21 893 99 (June) 1930
- Cullen T H A progressively enlarging ulcer of the abdominal wall invading

- Bowers W F : Appendicitis with especial reference to pathogenesis bacteriology and healing *Arch Surg* 39 302-422 (Sept) 1939
- Bowers W F and Richard Norman F Rectus muscle strain simulating acute appendicitis *Mil Surg* 92 645 8 (June) 1943
- Bowers W F and Shupe L Acute appendicitis sequela of typhoid inoculation *Mil Surg* 90 413 (Apr) 1942
- Boyce Frederick F *The Role of the Liver in Surgery* Charles C Thomas Springfield and Baltimore 1941
- Boyd William *Surgical Pathology* 4th ed W B Saunders Company Philadelphia and London 1938
- Brenneminn Joseph The abdominal pain of throat infections *Am J Dis Child* 22 493 11 (Nov) 1921
- Brennemann Joseph The abdominal pain of throat infections in children and appendicitis *J A M A* 89 2183 6 (24 Dec) 1927
- Brentano C Influence of insulin on glycogen formation from dextrose in normal animals *Klin Wchnschr* 18 42 6 (14 Jan) 1939 Abstract *J A M A* 112 1301 (1 Apr) 1939
- Breslin F J Gas bacillus infection of the abdominal wall in appendicitis *Am J Surg* 49 501 2 (Sept) 1940
- Bruce G G Diagnosis and treatment of acute appendicitis in children A review of 467 consecutive cases *Lancet* 1 1247 51 (3 June) 1939
- Bruhn Harold Acute pelvic appendicitis *Surg Gynec & Obst* 63 593 9 (Nov) 1936
- Bryant J D Removal of the vermiform appendix Letter from New York (Domestic Correspondence) *J A M A* 8 55 6 (8 Jan) 1887
- Burge R E Dennis C Varco H L and Wangenstein Owen H Histology of experimental appendical obstruction (rabbit ape and man) *Arch Path* 30 481 503 (Aug) 1940
- Bullowa J G M McCabe E J and Wishik S M Acute appendicitis in the exanthems *Am J Dis Child* 53 1029-38 (Apr) 1937
- Bumm E Zur Klinik der Greisenappendicitis *Deutsche Ztschr f Chr* 234 794 9 1931
- Bunch George H Mucoid disease of the appendix *Ann Surg* 121 704 9 (May) 1945
- Bunch George H and Adcock D F Grit faceted calculus of the appendix *Ann Surg* 109 143 6 (Jan) 1939
- Burford G E Pulmonary complications following 1 333 administrations of cyclopropane *J A M A* 110 1087 92 (2 Apr) 1938
- Burgess C M Traumatic appendicitis *J A M A* 111 699 700 (20 Aug) 1938
- Butler Stuyvesant Feenev Neil and Levine S A The patient with heart disease as a surgical risk Review of four hundred and fourteen cases *J A M A* 95 85 91 (12 July) 1930
- Butsch Winfield L and Harberson James C Acute virus infection with nerve root involvement simulating appendicitis *J A M A* 123 405 7 (16 Oct) 1943
- Cantor M O Kennedy O E and Reynolds H P Use and abuse of intestinal decompression tube A study based on 200 cases *Am J Surg* 73 437 49 (Apr) 1947
- Carlson H A and Wilder Lucretia The Schilling hemogram in appendicitis *Arch Surg* 30 325-35 (Feb) 1935
- Carlsaw R B On the character significance and prognostic value of peritoneal exudates *Brit J Surg* 3 8 35 (July) 1915
- Carter B N Left subphrenic abscess *Ann Surg* 110 562 77 (Oct) 1939

- Chenoweth Arthur I Appendical abscess *Surgery* 14 702 7 (Nov) 1943
- Christopher Frederick and Jennings W K Certain factors in the operative mortality of acute appendicitis *Am J Surg* 18 16 18 (Oct) 1932
- Clado Appendice caecal anatomie embryologie anatomie comparee bacteriologie normale et pathologique *Compt rend Soc de biol* 44 133 72 1892
- Coller F A and Brinkman H Studies on the reaction of the peritoneum to trauma and infection *Ann Surg* 109 942 54 (June) 1939
- Coller F A Campbell Kenneth E Vaughan Herbert F Lob L Vivian and Moyer Carl A Postoperative salt intolerance *Ann Surg* 119 533-42 (Apr) 1944
- Coller F A Dick V E and Maddock W G Maintenance of normal water exchange with intravenous fluids *J A M A* 107 1522 7 (7 Nov) 1936
- Coller F A Lob L Vivian Vaughan H H Kalder N H and Moyer C A Translocation of fluid produced by the intravenous administration of isotonic salt solutions in man postoperatively *Ann Surg* 122 663 77 (Oct) 1945
- Coller F A and Potter E B The treatment of peritonitis associated with appendicitis *J A M A* 103 1753-8 (8 Dec) 1934
- Collins D C Acute retrocecal appendicitis based on seven hundred and fifty one instances *Arch Surg* 36 729-43 (May) 1938
- Collins D C Diverticula of the vermiform appendix A study based on thirty cases *Ann Surg* 104 1001 12 (Dec) 1936
- Collins D C Etiological factors in acute appendicitis based upon a study of 3 400 cases *Surgery* 5 267 70 (Feb) 1939
- Collins D C Hematuria associated with acute appendicitis *Urol & Cutan Rev* 42 22-4 (Jan) 1938
- Collins D C Mechanism and significance of obliteration of the lumen of the vermiform appendix *Ann Surg* 104 199 211 (Aug) 1936
- Collins D C The treatment of complicated acute appendicitis *M Rec* 150 127 9 (16 Aug) 1939
- Colp Ralph Chills in acute appendicitis an analysis of 2841 cases of acute appendicitis treated in Mt Sinai Hospital of New York City *Ann Surg* 85 257 62 (Feb) 1927
- Comroe B I Non surgical causes of acute abdominal pain *Ann Surg* 101 438 44 (Jan) 1935
- Connell J E A Trauma and appendicitis *Surgery* 7 47 60 (Jan) 1940
- Cope Zachary *Clinical Researches in Acute Abdominal Disease* 2nd ed Oxford University Press London and New York 1927
- Cope Zachary *The Early Diagnosis of the Acute Abdomen* 9th ed Oxford University Press London and New York 1946
- Cope Zachary The prevention and early diagnosis of the acute abdomen *Brit M J* 1 6 9 (5 Jan) 1929
- Cope Zachary *The Treatment of the Acute Abdomen Operative and Post Operative* 2nd ed Oxford University Press London and New York 1928
- Copland S M and Mangolarra C J The mimicry of acute appendicitis by malaria *Rev Gastroenterol* 1 183 91 (Sept) 1934
- Corcoran William J Prenatal rupture of the appendix *Am J Dis Child* 39 277 80 (Feb) 1930
- Cosgrove S A Surgical complications of pregnancy *Am J Obst & Gynec* 34 469 79 (Sept) 1937
- Crisle G Jr Peritonitis of appendiceal origin treated with massive doses of penicillin Report of 50 cases *Surg Gynec & Obst* 83 150-62 (Aug) 1946
- Crocker W J and Valentine E H Hemography in the diagnosis of appendicitis based on 500 cases *J Lab & Clin Med* 21 893 99 (June) 1936
- Cullen T E A progressively enlarging ulcer of the abdominal wall invading

the skin and fat following drainage of an abdominal abscess apparently of appendiceal origin *Surg Gynec & Obst* 38 579 82 (May) 1924

Cullen T M and Brodel Max Lesions of the rectus abdominis muscle simulating an acute intra-abdominal condition *Bull Johns Hopkins Hosp* 61 29-348 (Nov) 1937

Curreri A R and Melick D W Foreign body in the appendix *Wisconsin M J* 40 192-3 (Mar) 1941

Cutler Condict W Urgent surgery in the aged *Ann Surg* 126 763 79 (Nov) 1947

Darrah L W Congenital absence of the vermiform appendix in a patient with mental disease *New England J Med* 214 776 9 (16 Apr) 1936

Davis G G A transverse incision for the removal of the appendix *Ann Surg* 43 106-10 (Jan) 1906

Dawson G R Appendicitis following tonsillitis and respiratory infections *Mil Surgeon* 80 447 55 (June) 1937

Deaver John Blair *Appendicitis Its History Anatomy Clinical Actiology Pathology Symptomatology Diagnosis Prognosis Treatment Technic of Operation Complications and Sequels* 4th ed P Blakiston's Son and Company Philadelphia 1913

DeBakey Michael E Phlebothrombosis and thrombophlebitis An analysis of 332 cases from Charity Hospital of Louisiana at New Orleans Personal communication

DeBakey Michael E and Polaski Edwin J An analysis of the experience with streptomycin therapy in United States Army hospitals Preliminary report *Surgery* 20 749 60 (Dec) 1946

DeCoursey J L Care of the ruptured appendix Reduction of mortality in four per cent in series of fifty cases *Surg Gynec & Obst* 63 756-60 (Dec) 1936

Dees J G A valuable adjunct in perforated appendices *Mississippi Doctor* 18 215 1<sup>st</sup> (Sept) 1940

Dennis C Burge R E Vairo R L and Wingensteen O H Studies in the etiology of acute appendicitis An inquiry into the factors involved in the development of acute appendicitis following experimental obstruction of the appendiceal lumen of the rabbit *Arch Surg* 40 929 48 (May) 1940

Deyle V F and Wallace J B Development of aplastic anemia during the use of streptomycin Report of two cases *J A M A* 136 1098 (24 April) 1948

Dixon C F and Deuterman J L The management of external intestinal fistulas *J A M A* 111 2095 2101 (3 Dec) 1933

Dorrance G M and Nealon S W Jr The use of cecal drainage in ruptured appendicitis *New York State J Med* 35 119 20 (1 Feb) 1935

Downs T McK Congenital malformations of the appendix—a familial disease *Ann Surg* 115 21 4 (Jan) 1942

Dvuzhlnaya E D Pathologico-anatomical changes in adjoining organs and tissues in acute appendicitis *Vestnik Khir* 61 59 67 (Jan) 1941 Abstract *Internat Abstr Surg in Surg Gynec & Obst* 73 537 8 (Dec) 1941

Earl J R BB shot in the vermiform appendix *J A M A* 114 1864 (11 May) 1940

Easton E R Unusual condition simulating acute appendicitis—Vincent's angina *Am J Surg* 22 74 7 (Oct) 1933

Edebohl G M The hernia guarantee and the minimum of confinement after operations for appendicitis with and without pus *M Rec* 55 665 7 (13 May) 1899

Eliason E L Early diagnosis in abdominal surgery *Am J Surg* 31 270 84 (Feb) 1936

Eliason E L Erb W H and Gilbert P D The *Clostridium welchii* and associated organisms A review and report of 43 new cases *Surg Gynec & Obst* 64 1005 14 (June) 1937

- Elliot J W A modification of the McBurney incision for appendectomy *Boston M & S J* 135 433-4 (29 Oct) 1896
- Elman Robert The challenge of acute appendicitis Diagnostic and therapeutic details designed to reduce mortality with special reference to the delayed operation *J Missouri State M A* 38 107 10 (Apr) 1941
- Elman Robert *Parenteral Alimentation in Surgery with Special Reference to Proteins and Amino Acids* Paul B Hoeber Inc New York and London 1947
- Fauley G B Duggan T L Stormont H T and Pfeiffer C C The use of penicillin in the treatment of peritonitis An experimental study *J A M A* 126 1132-4 (30 Dec) 1944
- Faxon H H Subphrenic abscess A report of one hundred and eleven consecutive operative cases *New England J Med* 222 299 99 (22 Feb) 1940
- Felson Benjamin and Bernhard C Melvin The roentgenologic diagnosis of appendiceal calculi *Radiology* 49 178 91 (Aug) 1947
- Fine Jacob Hermanson L and Frehling E Further clinical experiences with 95 per cent oxygen for absorption of air from the body tissues *Ann Surg* 107 1 13 (Jan) 1938
- Finney J M T The mimicry of the clinical picture of certain extra abdominal conditions by others of intra abdominal origin *South M J* 25 6 12 (Jan) 1932
- Finney J M T Jr Appendicitis Some observations based on a review of three thousand nine hundred thirteen operative cases *Surg Gynec & Obst* 56 360-65 (15 Feb) 1933
- Fitz Reginald The challenge of appendicitis *Ann Int Med* 12 1442 8 (Mar) 1939
- Fitz Reginald Concerning interns and their health *J A M A* 117 1125-30 (27 Sept) 1941
- Fitz Reginald Perforating inflammation of the vermiform appendix with special reference to its early diagnosis and treatment *Tr A Am Phys* 1 107-44 1886
- Flatley Gerald Torsion of the vermiform appendix associated with pregnancy *Lancet* 1 1357 (13 June) 1936
- Flynn G W Retroperitoneal approach in subphrenic abscess *Am J Surg* 17 163-8 (Aug) 1932
- Fossen A and Boeckle H E Frequency of acute appendicitis in Netherland East Indies *Geneeskundig tijdschr v Nederl Indie* 79 2857 67 (7 Nov) 1939
- Abstract *J A M A* 114 533 (10 Feb) 1940
- Foster Allan King Jr Acute mesenteric lymphadenitis simulating acute appendicitis *S Clin North America* 19 307 15 (Apr) 1939
- Foster Miles E and Bowers Warner F Obstructive appendicitis caused by a sprouting seed *Arch Path* 35 598 600 (Apr) 1943
- Fowler G R Diffuse septic peritonitis with special reference to a new method of treatment namely the elevated head and trunk posture to facilitate drainage into the pelvis with a report of nine consecutive cases of recovery *M Rec* 57 617 23 (14 Apr) 1900
- Fowler R H The rare incidence of acute appendicitis resulting from external trauma *Ann Surg* 107 529-39 (Apr) 1933
- Frazer W P Appendicitis and children—report of a case with intestinal parasite *Virginia M Month* 64 31-3 (Apr) 1937
- Galbreath W R and Irwin F G Appendicitis in Puerto Rico Observations based on a critical analysis of 244 cases *Am J Surg* 30 493 9 (Dec) 1935
- Gamm Kenneth Penicillin therapy in pyelophlebitis *J A M A* 128 1159 61 (18 Aug) 1945
- Gardner C E Jr Delayed operation in the treatment of the perforated appendix *Surgery* 4 161 78 (Aug) 1938
- Catch W D Cerv H E and Ballenger Felix Management of advanced

- appendicitis in childhood with observations on blood concentration due to peritonitis *J A M A* 114 1839-42 (11 May) 1940
- Gatewood John W Solitary diverticulitis of the cecum Report of two cases *Ann Surg* 122 52 8 (July) 1945
- Giertz K H Twenty five years experience in the treatment of peritonitis *Ann Surg* 104 712 35 (Oct) 1936
- Gilje Louis E and Lampson H Starr Acute appendicitis in amebic dysentery Report of a case *U S Nav M Bull* 46 169 11 (Jan) 1946
- Goodyear E S Traumatic appendicitis (Correspondence) *J A M A* 111 1788 (5 Nov) 1938
- Gordon S H Welchii infection complicating conservatively treated appendiceal abscess *Brit J Surg* 23 399-401 (Oct) 1936
- Graham H R and Brown W E Spinal anesthesia in abdominal surgery *Ann Surg* 110 863 71 (Nov) 1939
- Graham Wallace H Diagnosis of appendicitis with gastro enteritis *Mil Surgeon* 95 296-300 (Oct) 1944
- Grittan J F Gangrenous appendicitis coincident with labor An unusual case of initial attack occurring in and operated upon during first stage of full term labor in primipara Delivery Recovery *Surg Gynec & Obst* 29 457 61 (Nov) 1919
- Gray Henry Physical factors in the production of appendicitis Points in diagnosis Their influence on surgical treatment *Illinois M J* 48 33-8 (July) 1925
- Gray S H and Heifetz C J Lymphoid hyperplasia of the appendix with a note on its role in acute appendicitis *Arch Surg* 35 887 900 (Nov) 1937
- Green Howard W and Watkins Ralph M Appendicitis in Cleveland Final report *Surg Gynec & Obst* 83 613 624 (Nov) 1946
- Grette S Posttraumatic disturbances in region of appendix so-called traumatic appendicitis *Act chir Scandinav* 82 11 56 (13 Feb) 1939 Abstract *J A M A* 112 2105 (20 May) 1939
- Griffin Wilham D Silverstein Joseph Hardt Harry C and Seed Linton Prophylactic chemotherapy in appendicitis Preliminary report of one hundred and eight cases *J A M A* 133 907 9 (29 Mar) 1947
- Guerry LeG A study of the mortality in appendicitis *Ann Surg* 84 233 7 (Aug) 1925
- Haddon J W L A case of left sided appendicitis *Brit M J* 2 569 (27 Oct) 1945
- Hamilton Thomas Carcinoid tumour of the appendix causing acute appendicitis *M J Australia* 2 1086 7 (18 Dec) 1937
- Hamzah N A Amebic appendicitis *J Palestine Arab M A* 1 72-4 (Mar) 1946
- Handley W M Paralytic ileus in acute appendicitis *Proc Roy Soc Med* 29 163 70 (Nov) 1935
- Hardy James D and Godfrey Lincoln Effect of intravenous fluids on dehydrated patients and on normal subjects Cardiac output stroke volume pulse rate and blood pressure *J A M A* 126 23 5 (2 Sept) 1944
- Harrington F B Choice of method of opening the abdomen in appendicitis *Boston M & S J* 152 342 (23 Mar) 1905
- Harris Franklin I A new rapid method of intubation with the Miller Abbott tube *J A M A* 125 784 5 (15 July) 1944
- Harris Franklin I Intestinal intubation in bowel obstruction Technique with a new single lumen mercury weighted tube *Surg Gynec & Obst* 81 671 8 (Dec) 1945
- Harris Franklin I Diller Theodore and Marcus Sanford A Hemorrhagic infarction of the greater omentum simulating acute appendicitis *Surgery* 23 206 10 (Feb) 1948

Harris Franklin I and Gordon Milton Intestinal intubation in small bowel distention and obstruction Further experiences with the single lumen mercury weighted tube and analysis of complications *Surg Gynec & Obst* 86 647-58 (June) 1948

Harris W H Personal communication

Harris W H and Browne D C Ovarian vermicularis as a causative factor in appendicitis *J A M A* 84 650-54 (28 Feb) 1925

Harvey Harold D and Meleney Frank L Peritonitis A collective review of the significant literature for six and one half years *Internat Abstr Surg in Surg Gynec & Obst* 67 339-55 (Oct) 1939

Hawkes M Z Thrombophlebitis of appendical vein complicating acute appendicitis *Surg Gynec & Obst* 66 62-78 (Jan) 1938

Hays Albert T The treatment of acute appendicitis in a closed city *Surgery* 21 297-308 (Mar) 1947

Hextley Thomas F Hemorrhagic mucocele of the appendix *J A M A* 112 1935-6 (13 May) 1939

Henderson Yandell Atelectasis massive collapse and related post-operative conditions Wesley M Carpenter Lecture *Bull New York Acad Med* 11 639-56 (Nov) 1935

Herrera Rodolfo E Millet John H and Lawrence George H A report on the use of the Harris tube *Surg Gynec & Obst* 85 604-6 (Nov) 1947

Hertzler A E An inquiry into the nature of chronic appendicitis *Am J Obst & Gynec* 11 155-70 (Feb) 1926

Heyd C G Death from appendicitis A preventable mortality *Am J Surg* 28 317-20 (Nov) 1934

Hicken N F and Carlquist J H Primary appendical abscesses *Arch Surg* 42 156-71 (Jan) 1941

Hill W B and Mason C C Prenatal appendicitis with rupture and death *Am J Dis Child* 29 86-7 (Jan) 1925

Hinman E H Clinical aspects of Strongyloides stercoralis infection *Rev Gas troenterol* 24-34 (Mar) 1935

Hirshfeld J W Buggs C W Pilling M A Bronstein H and O'Donnell C H Streptomycin in the treatment of surgical infections report of experiences with its use *Arch Surg* 52 397-401 (Apr) 1946

Hochberg Lew A Subphrenic abscess A review of one hundred and eleven cases and a resumé of the subject *Arch Surg* 86 111-35 (Jan) 1938

Hogan E P The appendix problem A perennial cause of preventable mortality *Ann Surg* 105 815-44 (May) 1937

Holman C C Acute appendicitis treated by operation A record of twelve hundred cases *Lancet* 2 126-7 (16 July) 1938

Homans John *Circulatory Diseases of the Extremities* The Macmillan Company New York 1939

Howard R N Carcinoma of the appendix associated with acute appendicitis *M J Australia* 1 933-4 (28 May) 1935

Howes E L and Syphax Burke Successful removal of a gangrenous appendix in a premature newborn *Am J Surg* 50 133-9 (Oct) 1940

Hudson Henry W Jr and Chamberlain J W Acute appendicitis in childhood A statistical study of 848 cases from the Children's Hospital Boston *J Pediat* 15 408-25 (Sept) 1939

Hudson Henry W Jr and Krakower Cecil Acute appendicitis and measles *New England J Med* 215 59-64 (9 July) 1936

Hunnicutt Thomas Graf William J Hanburger Morton Ferris Eugene H and Scheinker I Mark Fatal toxic encephalopathy apparently caused by streptomycin *J A M A* 137 599-602 (12 June) 1948



- Hurwitt Elliott E Acute appendicitis occurring during the course of other diseases *New England J Med* 230 20 23 (2 Jan) 1947
- Jackson H C and Collier F A The use of sulfanilamide in the peritoneum Experimental and clinical observations *J A M A* 118 194 200 (17 Jan) 1942
- Jacobson Seymour S Childhood appendicitis *Am J Dis Child* 63 1110 22 (June) 1942
- Jemerin Edward E Postappendectomy incisional interstitial inguinal hernia *Surgery* 22 852 5 (Nov) 1947
- Jennings J E The relation of the Welch bacillus to appendicitis and its complications *Ann Surg* 93 828 36 (Apr) 1931
- Jennings J E Burger H H and Jacoba Mendel Acute appendicitis A clinical and pathologic study of 1090 consecutive cases *Arch Surg* 41 890-911 (May) 1942
- Jervis E Appendicitis during pregnancy and the puerperium *Acta obst et Gynec Scandinav supp* 4 1925 Abstract *Internat Abstr Surg in Surg Gynec & Obst* 43 311 12 (Oct) 1926
- Jirka F J and Scuderi C S Mucocysts of the appendix *Illinois M J* 73 57-8 (Jan) 1939
- John H W Chronic duodenal ulcer in a child of six years with fatal hemorrhage after appendectomy *Lancet* 1 433-4 (19 Feb) 1939
- Johns F S Appendicitis in middle aged Virginia M Month 5 509 11 (Nov) 1930
- Johnson A S The reliability of the leukocyte count in the diagnosis of appendicitis *New England J Med* 223 373 6 (5 Sept) 1910
- Johnson W W Gas gangrene infections following appendectomy *Am J Surg* 33 141 7 (July) 1936
- Jorpes J Erik Anticoagulant therapy in thrombosis *Surg Gynec & Obst* 84 677 81 (15 Apr) 1947
- Judd E S Snell A M and Hoerner M T Transfusion for jaundiced patients *J A M A* 105 1653 8 (23 Nov) 1935
- Kaufman Louis R Scipico Sibus and Mersheimer Walter A rare complication of the Miller Abbott tube *Am J Surg* 57 173 6 (July) 1942
- Kay John H and Lockwood John S Experimental appendicular peritonitis I The prognostic significance of certain hematologic factors especially the prothrombin time *Surgery* 20 56 71 (July) 1946
- Kay John H and Lockwood John S Experimental appendicular peritonitis II The significance of imbalance of circulating fibrinolytic and antifibrinolytic factor in the course of the disease *Surgery* 21 155 67 (Feb) 1947
- Kellogg W A Vesico intestinal fistula *Am J Surg* 41 136 86 (July) 1939
- Kelly F R and Watkins R M Appendicitis in adults Review of 2000 consecutive cases with comments on the rising mortality of the acute suppurative type *J A M A* 112 1795 8 (6 Mar) 1939
- Kelly H A and Hurdon E *The Vermiform Appendix and Its Diseases* W B Saunders and Company Philadelphia 1903
- Kelly J F and Dowell D A *Röntgen Treatment of Infections* The Year Book Publishers Inc Chicago 1942
- Kelly J F and Dowell D A The X ray treatment of acute peritonitis *Radiology* 39 299 306 (Mar) 1942
- Kessler H H Traumatic appendicitis *Am J Surg* 42 555 60 (Dec) 1938
- Keyes E L Death from appendicitis The mortality from appendicitis and the causes of death following appendicitis *Ann Surg* 99 47 69 (Jan) 1934
- Keyes E L Jr The sensation of gas stoppage during the onset of acute appendicitis *Surgery* 17 270 83 (Feb) 1945

- ✓ Keyes E L and Cook M M Diagnosis of acute appendicitis in the presence of diarrhea *Arch Surg* 52 429-44 (Apr) 1946
- Kitchen G H Haematemesis following appendectomy *Canad M A J* 36 357-9 (Apr) 1937
- Koop C Everett The management of parenteral fluids in geriatrics *Geriatrics* 1 269-76 (July Aug) 1946
- Koster H Thorium dioxide as an aid in the differential diagnosis of pylephlebitis *Radiology* 35 728-34 (Dec) 1940
- Kroger W P Tetanus following acute appendicitis *Am J Surg* 18 99-98 (sic) (Oct) 1932
- Lackner J E and Tubky A S Abortion as a complication of operation in the pregnant woman A plea for the prophylactic use of progesterone *Am J Surg* 46 362-4 (Nov) 1939
- Ladd William E Immediate or deferred surgery for general peritonitis associated with appendicitis in children *New England J Med* 219 329-33 (8 Sept) 1938
- Lam Conrad H and Hooker Donald H Pulmonary embolism A statistical study with particular reference to the value of certain preventive measures *Ann Surg* 123 221-8 (Feb) 1946
- ✓ Lamb C A Recurrent appendicitis *New England J Med* 219 746-8 (10 Nov) 1938
- Langmann A G Acute appendicitis and pseudoappendicitis in rheumatic children *J Pediat* 18 599-616 (May) 1941
- Langman T H and Ingalls T H Vitamin C deficiency and wound healing An experimental and clinical study *Ann Surg* 105 616-25 (Apr) 1937
- Lanz Der McBurney'sche Punkt *Zentralbl f Chir* 35 185-90 1908
- Latimer E O Mucocele of the appendix *Am J Surg* 47 124-7 (Jan) 1940
- Lazarus D Appendicitis in the aged *M Rec* 144 410-12 (4 Nov) 1936
- Leckie G G An interesting family history of appendicitis *Canad M A J* 36 287 (Mar) 1937
- Lehman E P and Parker W H The treatment of intraperitoneal abscess arising from appendicitis *Ann Surg* 108 833-56 (Nov) 1938
- Lehmann Hans Die Appendicitis acuta im Greisenalter *Wien klin Wchnschr* 40 995-7 (4 Aug) 1927
- Leithauser D J and Bergo H L Early rising and ambulatory activity after operation A means of preventing complications *Arch Surg* 42 1086-93 (June) 1941
- Lennander K G Ueber den Bauchschnitt durch eine Rectusscheide mit Verschiebung des medialen oder lateralen Randes des Musculus rectus *Centralbl f Chir* 25 90-94 (29 Jan) 1898
- Lennon F J Gall stones in the appendix Case report *New York State J Med* 38 1557 (15 Oct) 1936
- Lesser A and Kaufman L H Acute appendicitis with jejunal intussusception and abdominal lymphadenitis occurring in a child immobilized in a spica plaster cast Report of a case *New York State J Med* 38 1285-6 (1 Oct) 1938
- Lesser A and Keshun J G Acute appendicitis with abdominal abscess involving an undescended abdominal testicle Report of a case *J Urol* 43 715-17 (May) 1940
- ✓ Levitas M S New diagnostic points in appendicitis A clinicoanatomic consideration of bilateral hyperalgesia *Arch Surg* 44 918-32 (May) 1942
- LeWald L T Complete transposition of the viscera A report of twenty nine cases with remarks on etiology *J A M A* 84 261-8 (24 Jan) 1925
- Lewis Dean and Firor W M Appendicitis in Christopher F *A Textbook of Surgery by American Authors* W B Saunders Company Philadelphia and London 1936 pp 1184-97

Lewis Dorn and Penick R M Jr Fecal fistulae *Internat Clin* 1 (series 43) 111-30 (Mar) 1933

Liedberg Nils "Zur Frage der postoperativen fortschreitenden Hautgangrän nach Eingriffen in Bauch und Thorax" *Acta chir Scandinav* 77 351-77 (Oct) 1938

✓ Lintgen C and Fry K An evaluation of the sedimentation test in the differential diagnosis of acute pelvic inflammatory disease and acute appendicitis *Am J Obst & Gynec* 36 393-9 (Sept) 1939

Lipschutz H and Schifer C S Emergency abdomen: some observations of its incidence in persons older than age 50 *Pennsylvania M J* 38 5-9 (Oct) 1934

Loxton S Arthur Chassin James L and Hinton J William "Tissue protein depletion A factor in wound disruption" *Surg Gynec & Obst* 80 107-13 (Jan) 1918

Long LeRoy Downing "The prevention and treatment of postoperative intestinal incompetence Paralytic ileus" *South M J* 26 350-53 (Apr) 1933

Louvet J Bodart et Licoart Un cas de rupture silencieuse mort par péritonite appendiculaire *Bull Soc d'obst et de gynec* 23 371-3 (May) 1934

Love R J McN On the treatment of some acute abdominal disorders (Hunters Lecture) *Lancet* 1 375-81 (23 Feb) 1929

Loveland J E A lowered death rate for acute appendicitis *Am J Surg* 31 87-91 (Jan) 1936

Loveland J E Reginald Heber Fitz the exponent of appendicitis *Yale J Biol & Med* 9 509-20 (July) 1937

Ludington N A Factors in the etiology of traumatic appendicitis with certain clinical observations *J A M A* 80 1148-51 (19 May) 1923

✓ Lyons Champ Correlation of the use of antibiotic and chemotherapeutic agents with general principles of surgery *Surg Gynec & Obst* 84 729-32 (15 Apr) 1947

McArthur S W "The muscle splitting or grid iron incision for appendectomy" *Surg Gynec & Obst* 85 715-18 (Nov) 1937

McBurney Charles Experience with early operative interference in cases of disease of the vermiform appendix *New York M J* 50 676-84 (21 Dec) 1889

McBurney Charles "The incision made in the abdominal wall in cases of appendicitis with a description of a new method of operating" *Ann Surg* 20 39-43 (July) 1894

McBurney Charles Septic peritonitis following perforation of the vermiform appendix *New York M J* 47 719-21 (30 June) 1888

✓ McClure R D and Altemeier W A Acute perforated appendicitis with peritonitis Report of two hundred fifty two consecutive cases *Ann Surg* 103 800-814 (May) 1937

McClure W B Effect of acute appendicitis and of lobar pneumonia on the thoracic and abdominal respiratory movements in children *Am J Dis Child* 52 1047-60 (Nov) 1936

McDonald A L Appendicitis in pregnancy *Am J Obst & Gynec* 18 110-15 (July) 1929

McKenna Hugh Acute appendicitis in military hospitals (Correspondence) *J A M A* 116 532 (8 Feb) 1941

McKenna Hugh "The reduction of the increasing mortality and morbidity in acute appendicitis" *Ann Surg* 104 617-27 (Oct) 1936

McLanahan Samuel "Test for locating the position of the tip of the Miller Abbott tube" *J A M A* 138 35-6 (3 Jan) 1948

McLaughlin Edward F Ovarian lesions simulating appendicitis *Am J Surg* 57 115-21 (July) 1942

McNealy R W and Lichtenstein M H Acute mechanical ileal obstruction following appendectomy *Am J Surg* 55 157-61 (Jan) 1912

- McWhorter Golder L Subphrenic abscess An original extrapleural operation *Arch Surg* 35 241 57 (Aug) 1937
- Maddock W G and Collier F A Water balance in surgery *J A M A* 108 111 (2 Jan) 1937
- Mies U Boyce F F and McFetridge E M Post operative evisceration with an analysis of forty four cases *Ann Surg* 100 969 82 (Nov) 1934
- Mahorner H and Vincent R Optimum period for delay of operation following appendicitis complications *South Surgeon* 8 462 71 (Dec) 1939
- \* Manning J J Sign for acute retrocaecal appendicitis (Correspondence) *Brit M J* 2 770 (Dec) 1944
- \* Marbury W B Appendicitis in pregnancy *Am J Surg* 19 437-41 452 (Mar) 1933
- Mason M L Allen H S Queen F B and Gibbs E W A review of one thousand consecutive appendectomies *Quart Bull Northwest Univ M Sch* 15 1 20 1941
- ✓ Massie F M Amebiasis A cause of abdominal pain *South Surgeon* 9 584 92 (Aug) 1940
- ✓ Mastin E V Shoulder and clavicular pain in appendicitis *Ann Surg* 103 778 80 (May) 1936
- Matas R The surgical peculiarities of the American Negro a statistical inquiry based upon the records of the Charity Hospital of New Orleans *Tr Am S A* 14 483 610 (June) 1896
- \* Mayer Victor Acute appendicitis and pinworm infestation occurring concomitantly in the same family *J A M A* 115 1009 10 (21 Sept) 1940
- Mayo C W Appendicitis *Southwest Med* 18 397-403 (Dec) 1934
- Mayo C W and Schlicke C P The surgical management of fecal fistulae *Ann Surg* 114 1011 17 (Dec) 1941
- Meising Richard L Appendicitis complicating pregnancy labor and the puerperium *Surg Gynec & Obst* 85 512 22 (Oct) 1947
- Meleney Frank L A differential diagnosis between certain types of infectious gangrene of the skin With particular reference to haemolytic streptococcus gangrene and bacterial synergistic gangrene *Surg Gynec & Obst* 56 847 67 (May) 1933
- Meleney Frank L Zinc peroxide in surgical infections II *Clin North America* 16 691 711 (June) 1936
- Meleney Frank L Friedman Sidney T and Harvey Harold D The treatment of progressive bacterial synergistic gangrene with penicillin *Surgery* 18 422-35 (Oct) 1945
- Meleney Frank L Harvey Harold D and Jern H Z Peritonitis I The correlation of the bacteriology of the peritoneal exudate and the clinical course of the disease in one hundred and six cases of peritonitis *Arch Surg* 22 1 66 (Jan) 1931
- ✓ Millar W M Gas gangrene in civil life *Surg Gynec & Obst* 54 232-8 (1 Feb) 1932
- ✓ Miller E M Appendicitis in the aged *S Clin North America* 20 103-8 (Feb) 1940
- ✓ Miller T G The acute abdomen and the general practitioner *M Clin North America* 21 1095 1106 (July) 1937
- Moloney G E Acute appendicitis simulating gall stone ileus *Brit J Surg* 35 212 13 (Oct) 1947
- Monks G H Intestinal localization A review of certain studies (on the cadaver) in the surgical anatomy of the small intestine and its mesentery *Surg Gynec & Obst* 49 213 19 (Aug) 1929
- Moore Daniel C and Karp Mary Intravenous alcohol in the surgical patient *Surg Gynec & Obst* 80 523 5 (May) 1945

- Moore Matthew T. Patocysmal abdominal pain A form of focal symptomatic epilepsy. *J A M A* 124 561-3 (26 Feb) 1911
- Margison W J. Toxic reactions accompanying penicillin therapy. *J A M A* 132 915-19 (14 Dec) 1916
- Morley John. Abdominal pain as exemplified in acute appendicitis a clinical and biological consideration. *Brit M J* 1 85-90 (26 Mar) 1928
- Morrissey Paul G. Diagnosis of appendicitis in an IAP regional hospital. *Air Surgeons Bull* 2 184-5 (June) 1915
- Mortensen H N and Bray L. Case of appendicitis with unusual features. *M J Australia* 2 316 (15 Sept) 1928
- Movshian H G A. *Essays on Surgical Subjects Acute Appendicitis* W B Saunders Company Philadelphia 1921 pp 143-53
- Mueller Sterling. The local use of sulfanilamide in the treatment of acute appendicitis. A review of 1491 cases. *Ann Surg* 122 625-30 (Oct) 1945
- Mueller Sterling. The use of alcohol intravenously with special reference to its value in severe peritonitis. *S Clin North America* 19 401-5 (Apr) 1939
- Murphy John B. Appendicitis in pregnancy—Appendectomy. *Clinics of John B Murphy* 3 1055-1102 (Dec) 1914
- Murphy John B. Surgery of the appendix vermiformis Typhilitis perityphilitis epityphilitis. In Keen W W. *Surgery Its Principles and Practice* by Various Authors W B Saunders Company Philadelphia and London 1919 4 72-96
- Murphy John B. A talk on appendicitis. *Clinics of John B Murphy* 4 443-6 (June) 1915
- Murphy John B. A talk on a case of gangrenous appendicitis operated the previous evening. *Clinics of John B Murphy* 4 163-5 (Feb) 1915
- Murphy John B. Two thousand operations for appendicitis with deductions from his personal experience. *Am J M Sc* 125 187-211 (Aug) 1904
- Murphy John J. Ravdin Robert C and Zintel H A. The use of streptomycin in experimental peritonitis. *Surgery* 20 445-51 (Oct) 1946
- Murray Gordon. Anticoagulants in venous thrombosis and the prevention of pulmonary embolism. *Surg Gynec & Obst* 84 665-8 (15 Apr) 1947
- Murray Samuel D and Donelson Martin. Pylethrombophlebitis and hepatitis following appendectomy. Report of a case with recovery. *U S Nav M Bull* 45 1163-8 (Dec) 1945
- Nather K and Ochsner Alton. The left sided abdominal abscess as a complication of appendicitis. *Surg Gynec & Obst* 40 495-8 (Apr) 1925
- Nather K and Ochsner Alton. Retroperitoneal operation for subphrenic abscess with the report of two cases. *Surg Gynec & Obst* 37 665-73 (Nov) 1923
- \* Neiman Aaron. Acute appendicitis in diabetes mellitus. *Am J Surg* 49 443-8 (Sept) 1940
- Nelson Harry. Early ambulation following section of the anterior abdominal wall. An analysis of four hundred and twenty six personally conducted cases. *Arch Surg* 49 1-8 (July) 1944
- Nix James T and Nix James T Jr. Left sided appendicitis. A report of three cases. *New Orleans M & S J* 94 530-32 (May) 1942
- Norton J F and Connell J N. Appendicitis complicating pregnancy and labor. *Am J Surg* 32 325-32 (May) 1936
- Nuzum J W. Needless surgical operations from failure to recognize tubes der. *J A M A* 88 482-5 (12 Feb) 1916
- Ochsner A J. The cause of diffuse peritonitis complicating appendicitis and its prevention. *J A M A* 30 1747-54 (22 June) 1901
- Ochsner A J. A review of the histories of one thousand consecutive cases operated on at the Augustana Hospital during the thirty three months from July 1 1901 to April 1 1904. *South Med & Surg* 2 203-12 1904

Ochsner Alton Subphrenic abscess its diagnosis and treatment with special reference to the extraperitoneal operation *Internat Clin* 2 (series 41) 79 III (June) 1931

Ochsner Alton "The use of vasodilatation in the treatment of venous thrombosis" *Surg Gynec & Obst* 84 639-64 (15 Apr) 1947

Ochsner Alton and DeBakey Michael Subphrenic abscess Collective review and an analysis of 3603 collected and personal cases *Internat Abstr Surg* in *Surg Gynec & Obst* 66 428-38 (May) 1938

Ochsner Alton and DeBakey Michael Surgical consideration of amebiasis Collective review *Internat Abstr Surg* in *Surg Gynec & Obst* 69 392-403 (Oct) 1939

Ochsner Alton and DeBakey Michael "Therapeutic considerations of thrombophlebitis and phlebothrombosis" *New England J Med* 225 207-27 (7 Aug) 1941

Ochsner Alton Gage I M and Garside Earl "The intra abdominal post-operative complications of appendicitis" *Ann Surg* 91 544-72 (Apr) 1930

Ochsner Alton and Lilly George "The technique of appendectomy with particular reference to the treatment of the appendical stump" *Surgery* 11 532-54 (Oct) 1937

Orr T G "The action of morphine on the small intestine and its clinical application in the treatment of peritonitis and intestinal obstruction" *Ann Surg* 98 835-40 (Nov) 1933

Orr T G "Treatment of peritonitis" (Clinical Lecture at St Louis Session) *J A M A* 113 1489-92 (14 Oct) 1939

Ottenberg R and Berck M Sulfanilamide therapy for suppurative pylephlebitis and liver abscesses *J A M A* 111 1374-5 (8 Oct) 1938

Ottolenghi E L "Ulceration of external iliac vein and artery as rare postoperative complication of gangrenous appendicitis" *Revista de la Asoc Med Argentina* 42 427-504 (15-30 June) 1941 Abstract *J A M A* 117 1742 (15 Nov) 1941

Overholt R H and Donchess J C Subphrenic abscess *New England J Med* 213 294-301 (15 Aug) 1935

Pareira Morton D and Somogyi Michael Rationale of parenteral glucose feeding in the postoperative state *Ann Surg* 127 417-25 (Mar) 1948

Payne R L "The straight vessels of the terminal ileum" *Tr South S A* 34 346-7 (Dec) 1921

Pemberton J de J Pool T L and Miller J M Vesico-appendiceal fistulas *J Urol* 44 274-8 (Sept) 1940

Pendergrass E P and Hodes P J Acute postoperative parotitis *Radiology* 38 307-10 (Mar) 1942

\* Perry T and Keeler C E "Thirty nine cases of appendicitis in a single family pedigree" *Am J Surg* 46 259-65 (Nov) 1939

Petty M J Internal faecal fistula and death following appendicectomy *Brit M J* 11 491-2 (9 Sept) 1933

Plewes Burns Pulmonary embolism *Canad M A J* 41 271-4 (Sept) 1939

Plewes W F Abdominal pregnancy complicated by appendicitis and bilateral pyosalpinx *Canad M A J* 37 172-3 (Aug) 1937

Pool Eugene H Complete transposition of viscera in a case of diffuse appendiceal peritonitis *Ann Surg* 56 940-42 (Dec) 1912

Probstern J G and Lassar G N Mucocoele of the appendix with myxoglobulosis *Ann Surg* 127 171-6 (Jan) 1948

Pulaski Edwin J Seeley Sam F and Matthews Charles S Streptococci in surgical infections IV Peritonitis *Surgery* 22 889-99 (Dec) 1947

Quinn J S Acute appendicitis complicating uterine inertia *Irish J M Sc* 6 88-9 (Feb) 1938

- Rambo C M and Lasky L Congenital absence of the vermiform appendix Case reports *Ohio State M J* 34 394 (Apr) 1938
- Ravdin I S Some aspects of carbohydrate metabolism in hepatic disease *J A M A* 93 1193 9 (19 Oct) 1929
- Ravdin I S and North John P The simultaneous occurrence of acute appendicitis and malaria *Ann Surg* 122 432 5 (Sept) 1945
- Ravdin I S Stengel Alfred Jr and Prushankin M The control of hypoproteinemia in surgical patients *J A M A* 114 107 12 (13 Jan) 1940
- Ravdin I S Vars H M Goldschmidt S and Klingensmith L E Anesthesia and liver damage II The effect of anesthesia on the blood sugar the liver glycogen and liver fat *J Pharmacol & Exper Therap* 64 111 29 (Sept) 1938
- Ravdin I S Vars H M Thorogood E Schultz J and Johnson J The liver glycogen and lipid concentrations following intravenous glucose administration and diet in dog and man in the presence of liver damage *Ann Surg* 114 1018-25 (Dec) 1941
- Ray H S A study of appendicitis 1500 cases at the New York Hospital *New York State J Med* 38 412 23 (15 Mar) 1939
- Reid Mont R The appendicitis problem (Editorial) *Surgery* 3 601-5 (Apr) 1938
- Reid Mont R and Montanus W P Appendicitis An analysis of 1153 cases at the Cincinnati General Hospital *J A M A* 114 1307 11 (6 Apr) 1940
- Reid Mont R Poer D H and Merrell Paul A statistical study of 2921 cases of appendicitis *J A M A* 106 665 9 (29 Feb) 1936
- Reingold Irving M and Webb Frank R Sudden death following intravenous injection of thiamine hydrochloride *J A M A* 130 491 2 (23 Feb) 1946
- Rhoads J E Flugelman M T and Panzer L M The mechanism of delayed wound healing in the presence of hypoproteinemia *J A M A* 118 21-5 (3 Jan) 1942
- Riegel Cecilia Koop C E Grigger R P Rhoads J E and Bullitt L The protein requirements of surgical patients during the postoperative period *S Clin North America* 25 1096 1105 (Oct) 1945
- Ries F Some radical changes in the after treatment of celiotomy cases *J A M A* 33 454 6 (19 Aug) 1899
- Riggs T F The prone and right lateral position for gravity drainage in perforated appendicitis *Nebraska State M J* 22 365 70 (Oct) 1937
- Roberts Arwyn Carcinomatosis simulating appendicitis *Brit M J* 1 153 (3 Feb) 1945
- Robertson D E Appendix vermiformis duplex *Canad M A J* 43 159 61 (Aug) 1940
- Rockey E A Transverse incision in abdominal operations *M Rec* 68 779-80 (11 Nov) 1905
- Ronaldson G W Appendicular symptoms in the acute infectious diseases *Brit J Child Dis* 33 85 92 (Apr June) 1936
- Rose H W Severe burns complicated by appendicitis *Northwest Med* 36 118-14 (Apr) 1937
- Rosenow E C The bacteriology of appendicitis and its production by intravenous injection of streptococci and colon bacilli *J Infect Dis* 16 240 68 (Mar) 1915
- Rouhier Appendicite a la guerre Appendicite et fièvre typhoïde *Bull et Mem Soc de Chirur de Paris* 43 1846 50 (10 Oct) 1917
- Royster H A *Appendicitis* D Appleton and Company New York and London 1927
- Rubenstein A Daniel and Johnson Ben B Salmonella appendicitis *Am J M Sc* 210 517 23 (Oct) 1945

- Ryan W J Hernia of the vermiform appendix *Ann Surg* 106 135 9 (July) 1937
- Saphir O The anatomic diagnosis of acute appendicitis *Am J Clin Path* 11 163 9 (Feb) 1941
- Sauer II and Bailey F W Appendicitis *S Clin North America* 20 1261 79 (Oct) 1940
- Schenken J R and Moss Emma S Enterobius vermicularis in the appendix report of study on 1 000 surgically removed appendices *Am J Clin Path* 12 509 517 (Oct) 1942
- Schenken J R and Moss Emma S Intestinal parasitism in the Southern Negro *Tri State M J* 13 2690 91 (Feb) 1941
- Schullinger II N Acute appendicitis and associated lesions: Some observations on the mortality *Arch Surg* 32 65 98 (Jan) 1936
- Schulz Irwin Perforated appendix in children *Arch Surg* 56 117 22 (Jan) 1948
- Scott H William and Ware Paul F Acute appendicitis in childhood *Arch Surg* 50 258 68 (May) 1945
- Sherren J On the occurrence and significance of cutaneous hyperalgesia in appendicitis *Lancet* 2 816 21 (Sept) 1903
- Short A Rendle *The Causation of Appendicitis* John Wright and Sons Bristol 1946
- Shrager V L and Ragins A B Strangulated internal hernia simulating appendicitis *Am J Surg* 29 306-8 (Aug) 1935
- Silvani Henry L Rothenberg Sanford Warner Helen Amluxen Joyce and McCorkle H J Laboratory and clinical experiences with streptomycin therapy in the management of infections of intestinal origin *Surg Gynec & Obst* 78 721 8 (Dec) 1947
- Simeone F A and Stewart J D Acute hepatitis jaundice and abnormal bleeding as complications of acute appendicitis with perforation *New England J Med* 223 632-4 (17 Oct) 1940
- Simpson R H A common operative finding in appendicitis *J Missouri State M A* 38 128 9 (Apr) 1941
- Smith J A and Bartlett M K Acute surgical emergencies of the abdomen in pregnancy *New England J Med* 223 529 31 (3 Oct) 1940
- Smyth C M Jr The present status of the management of spreading peritonitis of appendiceal origin *Internat Clin* 4(ns 3) 273 90 (Dec) 1940
- Snyder W H Hall M G and Allen A W The association of pylephlebitis and appendicitis *New England J Med* 212 183 91 (31 Jan) 1935
- Stalker L K Appendicitis among individuals more than sixty years of age *Surg Gynec & Obst* 71 54 9 (July) 1940
- Steinberg Bernhard *Infections of the Peritoneum* Paul B Hoeber Inc New York 1944
- Steinberg Bernhard Peritoneal exudate A guide for the diagnosis and prognosis of peritoneal conditions *J A M A* 116 572 8 (15 Feb) 1941
- Steinberg Bernhard Stages in peritonitis based on the defense mechanism in relation to treatment *Arch Surg* 39 770 82 (Nov) 1939
- Steinberg Bernhard and Martin Ruth A Diffusion and localization of experimental infections of the peritoneum *Surg Gynec & Obst* 79 457 63 (Nov) 1944
- Stemer P E Necropsies on Okinawans Anatomic and pathologic observations *Arch Path* 42 359 80 (Oct) 1946
- Stevenson W O A case of appendicitis with most unusual symptoms *Canad M A J* 39 263-4 (Sept) 1938
- Stone C S Jr Acute appendicitis in children *Arch Surg* 30 346 56 (Feb) 1935



Stone Harvey B The management of acute appendicitis Arguments and controversies *Virginia M Month* 87 655 11 (Nov ) 1940

Strohl E Lee Appendectomy by the muscle splitting incision *S Clin North America* 22 1 8 (Feb ) 1942

Swartzwelder J C Clinical Trichocephalus trichiurus infection An analysis of eighty one cases *Am J Trop Med* 19 473 81 (Sept ) 1939

Tammann H and Lohmann H Acute appendicitis in the aged *Med Klin* 30 1235 6 (14 Sept ) 1934

✓ Tarnowsky George de Surgery of the aged indications and contraindications *S Clin North America* 20 3 12 (Feb ) 1940

Tiube H N A case of ulcerative colitis with unusual complications *Am J Digest Dis* 5 259 60 (June) 1938

Taylor I B Bennett J H and Waters R M Anesthesia at the Wisconsin General Hospital A three year statistical report Part I Anesthetic methods and postoperative respiratory complications *Anesth & Anal* 16 187 92 (Jul-Aug ) 1937

Tennison W J III and Dixon C F The relationship between fecaliths in the appendix and gangrenous appendicitis *South Surgeon* 10 111 13 (Feb ) 1941

Thalheimer William Chills occurring early in appendicitis before operation and their indication of an operable stage of pykphlebitis *Arch Surg* 8 655 68 (Mar ) 1924

Thompson J E Brabson J A and Walker J M The intra abdominal application of sulfanilamide in acute appendicitis *Surg Gynec & Obst* 72 722 7 (Apr ) 1941

Thompson W D Ravdin I S and Frank I L Effect of hypoproteinemia on wound disruption *Arch Surg* 38 500 503 (Mar ) 1939

Throckmorton T D The peritoneal response to powdered sulfonamide compounds An experimental study *Proc Staff Meet Mayo Clinic* 10 423 5 (2 July) 1941

Totten H P Mortality factors in appendicitis with perforation *Ann Surg* 106 1035 45 (Dec ) 1937

Touroff A S W Peritoneal aspiration as an aid in the diagnosis of acute appendicitis *S Clin North America* 19 287 94 (Apr ) 1939

Treves Frederick Lectures on the anatomy of the intestinal canal and peritoneum in man *Brit M J* 1 527-30 (14 Mar ) 1893

Turner G Grey Acute appendicitis *Brit M J* 2 691-5 (1 Oct ) 1938

Twyman R A Mussey R D and Stalker L K Appendicitis in pregnancy a review of seventy five cases *Proc Staff Meet Mayo Clin* 15 484 7 (31 July) 1940

Vale C F Postappendiceal abscess in the rectovesical pouch transrectal drainage *Ann Surg* 111 396-402 (Mar ) 1940

Vickers H D and Conrad J W Congenital pyloric stenosis in a premature infant followed by gangrenous appendicitis *Am J Surg* 49 503 9 (Sept ) 1940

Wikeley C P G Hernia of the vermiform appendix A record of sixteen personal cases *Lancet* 2 1282-4 (3 Dec ) 1938

Wikeley C P G Prognosis of appendicitis in childhood *Lancet* 2 319 -0 (11 Aug ) 1934

✓ Walker I J Immediate or deferred surgery for general peritonitis associated with appendicitis in adults *New England J Med* 219 323 9 (8 Sept ) 1938

Wangensteen O H The genesis of appendicitis in the light of the functional behavior of the vermiform appendix (Lewis Linn McArthur Lecture) *Proc Institute Med Chicago* 12 266 90 (15 Feb ) 1939

Wangensteen O H *Intestinal Obstructions a Physiological and Clinical Consideration with Emphasis on Therapy Including Description of Operative Procedures* 2nd ed Charles C Thomas Springfield 1942

- Wangensteen O H The mechanism of the vermiform appendix—a potential "closed loop" *Surg Gynec & Obst* 62 1020-22 (June) 1936
- Wangensteen O H and Bowers W F Significance of the obstructive factor in the genesis of acute appendicitis An experimental study *Arch Surg* 34 496-526 (Mar) 1937
- Wangensteen O H Burge R E Dennis C and Ritchie W P Studies in the etiology of acute appendicitis The significance of the structure and function of the vermiform appendix in the genesis of appendicitis A preliminary report *Ann Surg* 106 910-42 (Nov) 1937
- Wangensteen O H and Dennis C Experimental proof of the obstructive origin of appendicitis in man *Ann Surg* 110 629-47 (Oct) 1939
- Wangensteen O H and Dennis C The production of experimental acute appendicitis (with rupture) in higher apes by luminal obstruction *Surg Gynec & Obst* 70 799-806 (Apr) 1940
- Ward Robertson Appendicitis with complications A reduction in mortality due to use of continuous gastrointestinal decompression *West J Surg* 48 469-79 (Aug) 1940
- Warnock F B A comparative study of the leucocyte count and histopathology in acute appendicitis Value of the Schilling count in establishing the diagnosis *Am J Surg* 21 47-55 (July) 1933
- Watson Leigh F *Hernia Its Anatomy Etiology Symptoms Diagnosis Differential Diagnosis Prognosis and Operative Treatment* 2nd ed C V Mosby Company St Louis 1938
- Waugh T R Appendix veriformis duplex *Arch Surg* 42 311-20 (Feb) 1941
- Wechsler I S Abdominal pain as a symptom of disease of the brain *J A M A* 105 647-50 (31 Aug) 1935
- Weir H F A new use for the useless appendix in the surgical treatment of obstinate colitis *M Rec* 62 201-2 (9 Aug) 1902
- Welch C E and Faxon H H Thrombophlebitis and pulmonary embolism *J A M A* 117 1502-8 (1 Nov) 1941
- Wharton Lawrence R and Henriksen Erle Studies in ovulation The operative observations in periodic intermenstrual pain *J A M A* 107 1425-32 (31 Oct) 1936
- Wilkie D P D Acute appendicitis and acute appendicular obstruction *Brit M J* 2 959-62 (5 Dec) 1914
- Wilkie D P D Appendicitis *Ann Surg* 100 202-5 (July) 1934
- Wilkie D P D Observations on mortality in acute appendicular disease *Brit M J* 1 253-5 (14 Feb) 1931
- Wilkie D P D The prognostic value of an immediate examination of peritoneal exudates *Internat Clin* 4 145-52 1912
- Willis Murat The treatment of the appendix stump after appendectomy *Ann Surg* 48 74-9 (July) 1903
- Willson Pepper J K Impressions of surgery in West Africa *Brit M J* 2 812-14 (8 Dec) 1945
- Winslow S B Dextrose utilization in surgical patients *Surgery* 4 867-80 (Dec) 1938
- Wishart J H and Peterson L J Streptomycin therapy in pyothrombophlebitis *J A M A* 133 539-41 (22 Feb) 1947
- Wohl Michael G Metabolic disturbances simulating acute abdominal emergencies *S Clin North America* 26 1493-1506 (Dec) 1946
- Wood C B Acute appendicitis in the aged A study of 43 cases occurring after the age of 60 *Am J Surg* 26 321-5 (Nov) 1934

- Wood F C Radiology of the appendix (appendiculography) *Brit M J* 1 640 42 (30 Mar) 1935
- Woodruff R and McDonald J R Benign and malignant cystic tumors of the appendix *Surg Gynec & Obst* 71 750 55 (Dec) 1940
- Wright T Aaron A H Regan J S and Mulch E "The management of patients with diffuse peritonitis caused by perforation of the appendix" *J A M A* 113 1285 8 (30 Sept) 1939
- Wrook D H A study of abdominal rigidity *Proc Staff Meet Mayo Clin* 15 393 8 (19 June) 1940
- Yater Wallace M Diagnosis of liver abscess by means of thorotrast hepatosplenography *J A M A* 125 775 8 (15 July) 1944
- Yodanis Arnoldo A new sign to differentiate abdominal muscular rigidity in cases of acute abdominal conditions from that of other causes *Am J Surg* 57 457 8 (Sept) 1942
- Young John P Jr and Cole Warren H Intraperitoneal administration of succinylsulfathiazole and phthalylsulfathiazole Their use in the prophylaxis and treatment of peritonitis *Arch Surg* 53 182 9 (Aug) 1946
- Zahawi S Intestinal obstruction and atrophic lesion of the appendix caused by ascaris *J Trop Med & Hyg* 41 316 19 (1 Oct) 1938
- Zierold A A Morphine as a diagnostic agent *Proc Staff Meet Mayo Clin* 10 297-304 (8 May) 1935
- Zintel Harold A Flippin Harrison F Nichols Anna C Wiley Marjorie M and Rhoads J E Studies on streptomycin in man I Absorption distribution excretion and toxicity *Am J M Sc* 210 421-30 (Oct) 1945
- Van Zwilenburg C Obstruction and consequent distention the cause of appendicitis as proved by crises and by experimental appendicitis in dogs *J A M A* 42 820 27 (26 Mar) 1904
- Van Zwilenburg C The relation of mechanical distention to the etiology of appendicitis *Ann Surg* 41 43-50 (Mar) 1905

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